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THE MEDICAL CLINICS
OF
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CLINIC OF DR ARTHUR R ELLIOTT

ST LUKE'S HOSPITAL

SERIOUS EFFECTS OF EPISTAXIS IN CARDIOVASCULAR CONDITIONS

THAT severe bleeding from the nose may develop in individuals having high blood pressure is well known I bring the matter to your attention in order to emphasize the peculiar character of this development when it occurs along with hypertension and the possible serious effects which may follow if the hemorrhage be profuse or prove intractable The matter is fresh in my mind because of two occurrences, both of which proved disastrous One of these occurred in a patient who died on my service in this hospital just three weeks ago his death undoubtedly hastened by severe and repeated nosebleed A young man of twenty one was brought into the hospital in a state of profound anemia December 4th There was evidence of recent bleeding from the right nostril The account given by the patient and confirmed by members of his family was that for a period of four months he had had frequent epistaxis increasing gradually in severity During the six weeks preceding admission bleeding from the right nostril had occurred almost daily and on several occasions the loss of blood had been excessive and prolonged He had on three occasions particularly been greatly prostrated and had fainted several times There had occurred a loss of 15 pounds of body weight during the preceding month Four days prior to entry the hemorrhage, which, though not profuse, had proved intractable and prolonged, ceasing only

when the patient was on the verge of syncope and extremely pallid Immediately following admission to the ward a blood count was made, the red count yielding 1,260,000 erythrocytes, with hemoglobin 20 per cent and leukocytes 15,300 There appeared to be no air hunger or restlessness He was fully conscious and able to give an intelligent account of his recent experiences The rectal temperature was 99° F , pulse 90, regular and tense A review of the patient's past history revealed no incident likely to have a bearing on the intractable epistaxis except the interesting and possibly significant fact that in early youth he had been under medical care for several months on account of "kidney trouble" with albuminuria and dropsy After discharge from observation the urinary condition was never reinvestigated and his dropsy never recurred The surprising thing that immediately struck the attention was a decided tension and incompressibility of the radial pulse—not a thing to be expected in one so profoundly anemic The palpable arteries generally gave to the finger the impression of marked fibrosis The blood-pressure proved to be 178/80 His heart was found enlarged in its transverse diameter, the second aortic sound was sharply accentuated and there was a general blowing systolic murmur Ophthalmoscopic examination revealed a pale but normal fundus except for very evident fibrosis of the retinal arteries The urine was scant in amount, the specific gravity 1 014, plain reaction for albumin and in the sediment a few red cells and leukocytes and moderate number of casts

A diagnosis of chronic nephritis with hypertension and posthemorrhagic anemia was made Strict quiet was enforced, a sedative administered, and a glucose saline proctoclysis administered by slow drip

On the third day after entry epistaxis recurred, but yielded quickly to control, not, however, before the blood count had been still further reduced to 1,140,000 per cubic millimeter There is reason to believe that considerable blood had run backward into the pharynx and been swallowed, as the stool next day was heavily charged therewith The blood chemistry revealed high-

grade azotemia, urea nitrogen being 187 mg per cent, total non-protein nitrogen 328 mg, and blood creatinin 12 5 mg The blood Wassermann was reported negative

There was complete anorexia, so that it was difficult to introduce even fluids The patient slept a great deal and when awake appeared indifferent and stupid He complained of persistent nausea and vomited daily On the eighth day bleeding began again from the right nostril, and was reported by the rhinologist as coming from the area of Kiesselbach, much of the blood escaping into the pharynx and down the esophagus The area of bleeding was cauterized and the right nostril packed Seven hundred c.c of whole blood was given by transfusion from an appropriate donor Following this transfusion the patient's count rose to 1,500,000 and hemoglobin to 40 per cent Within a few hours after right nasal bleeding had been controlled a sharp hemorrhage began from the left nostril which, in turn, required packing His blood count now stood at 1 320,000 and his blood pressure 150/80 Epistaxis did not recur the next day after removal of packs from both nostrils, but the patient's toxemia deepened rapidly into coma with muscular twitching, slow labored breathing, and finally death in uremia, forty-eight hours after the last hemorrhage A few hours before death the total non protein blood-nitrogen reached 404 mg and CO_2 combining power 32 4 per cent Necropsy was refused

Consideration of the fragment of this patient's clinical history represented by the foregoing account of its terminal stage will, I think, convince one that chronic nephritis had existed for a long time, perhaps from early youth and possibly from the illness represented as "kidney trouble with albuminuria and dropsy" which occurred during that age period Well-established vascular and cardiac alterations existed as evidence of a serious constitutional disturbance operating throughout a lengthy period, and the high concentration of blood nitrogen derivatives testified to the advanced state of renal function impairment The progress of his nephritis had been characterized by such latency of symptoms as to leave him in ignorance of its existence until the onset of epistaxis That this came from a small artery there

can be no doubt Such severe repeated and intractable hemorrhage practically never comes from capillaries or veins within the nose, but from small arteries, the walls of which are undermined by disease The profound secondary anemia resulting from so much loss of blood by imposing a further handicap upon the kidneys as well as through its effects upon nutrition sufficed to hurry along the uremia and retention acidosis that terminated life As the event proved in this case, epistaxis constituted a complication which cut short what otherwise might have been a longer clinical history, although most certainly one that in the end must have terminated in uremia but by a more gradual course

An effect somewhat out of the ordinary resulting from the anemia induced by severe epistaxis is exemplified in another patient who was fifty years of age at the time of his death For four years this gentleman had been under my observation with elevated blood-pressure, the average range of which was about 180/115 He had a bad cardiovascular history on both sides of the house, his father and mother both having chronic vascular hypertension, the former dying of coronary thrombosis and the latter from cerebral apoplexy One year before his death this patient had his first attack of angina pectoris This was not repeated until the events about to be recorded, although during the interim he had many mild discomforts of anginal nature, especially if he overexerted or indulged in alcohol to excess, which he persisted in doing despite warnings The urinary findings and blood nitrogen were normal and the Wassermann test negative During September, 1928 he developed epistaxis After two preliminary slight and easily controlled nosebleeds he experienced a severe prolonged hemorrhage which required tamponing of both nostrils for control The loss of blood was so severe as to reduce his count to 3,000,000 and his hemoglobin to 50 per cent Almost immediately following this hemorrhage he had a severe angina and this recurred so frequently thereafter as to make his life miserable until his sudden death in December from coronary occlusion

Although he did not experience a second severe epistaxis

there occurred every few days slight bleeding from the nose lasting from a few minutes to half an hour, yielding to control by direct pressure This loss of blood sufficed to prevent a return of the count to normal since it never rose again above 3,500,000

In this case it appears likely that the anemia suddenly precipitated by severe loss of blood from nasal hemorrhage aggravated the angina and hastened the patient's death from coronary occlusion His previous history of high blood pressure for several years and angina extending over a year or more points to established vascular and myocardial damage That his coronary circuit must have been considerably narrowed by sclerosis seems evident enough in view of his substernal pains on exertion and following indiscretions Coronary ischemia easily induced by effort and excitement bespeaks a pretty narrow margin of reserve in the coronary circulation A depleted blood added to an already sluggish coronary flow might fairly enough, it seems to me, suffice to hurry along the fatal termination

In 1918 Herrick and Nuzum,[1] in an article on angina pectoris, called attention to the occasional association of angina pectoris and severe anemia They state that blood of poor quality going through somewhat narrowed coronaries might favor the development on slight provocation of an anginal spell They reported four patients with high grade anemia who had perfectly typical anginal attacks on exertion Herrick in 1927 reopened the subject for discussion, reporting three other similar cases and reviewing the literature[2] From this study Herrick concludes that there is occasionally seen a combination of angina pectoris with severe anemia The combination may, at times, be purely accidental At other times, though there is usually a groundwork for angina in cardiovascular lesions, the anemia is a factor contributing to the production of the painful seizures

In the case reported herewith the abrupt and persistent aggravation of the angina would appear to be more than mere coincidence. What part the anemia played in inducing the coronary occlusion is more of a conjecture

[1] Herrick and Nuzum Jour Amer Med Assoc. 70 67 1918
[2] Herrick, J B Trans Assoc. of Amer Phys vol xlii p 23

CLINIC OF DR LEWIS J POLLOCK

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THE NEUROLOGIC ASPECT OF OPTIC NEURITIS

THE synonymous use of the terms "optic neuritis," "choked disk," "papilledema" "papillitis," and "neuroretinitis" has led to such confusion of clinical interpretation that a careful analysis of the literature is almost impossible Even were we to accept a clear differentiation between papilledema, papillitis, and optic neuritis, the difficulties encountered in their diagnosis have as yet not been surmounted Although, perhaps relatively simple to the ophthalmologist, I am sure that most of the neurologists will agree that ophthalmoscopic examination does not permit of the ready differentiation of papillitis from papilledema It is difficult to determine whether a papilledema has been followed by a local toxic change producing a superimposed papillitis I have found it particularly difficult to determine whether we were dealing with an early papilledema or papillitis Similarly, to determine whether an optic atrophy was present in a subsiding papilledema, or whether the papilledema was superimposed upon an optic atrophy When one is not privileged to examine a patient at frequent intervals from the onset of disability, the presence of a papilledema complicated by the widespread changes of a superimposed papillitis or neuroretinitis offers many pitfalls in diagnosis

Many adjunctive signs are, of course, useful, visual acuity, size of blind spots, and perimetric fields frequently are of assistance, but just as often one is misled I need not stress at this time the well-known fact that visual acuity is preserved for a considerable time in the presence of a papilledema Nor is it necessary to call attention to the fact that spontaneous recession of a papilledema rarely occurs, and that where it does it is asso

ciated with syphilis, brain abscess, solitary tubercles of the brain, and tumor suspects, ordinarily classified as pseudotumor It is exceedingly likely that in many of these cases a papillitis was present

Illustrative of this, a child rather quickly developed a marked inco ordination of gait due to cerebellar dysfunction, at the same time a marked behavior disturbance was noted He became impatient, fretful, easily angered, markedly restless, and destructive, cried and shrieked Aside from cerebellar ataxia little was found upon neurologic examination A moderate degree of what appeared to me to be a papilledema was present The cerebrospinal fluid was essentially negative The cerebellar ataxia was so marked that a supratentorial tumor was suspected Because of the marked change in behavior it was felt that further observation of the ophthalmoscopic findings should be made, and if they receded that operation should be deferred Not only has the papilledema receded, but within a few weeks cerebellar ataxia has markedly diminished

Of course, one cannot make a positive diagnosis in this case as well as in the case of a patient of Dr Bernard Sachs of New York, where a boy, aged nine years, presented many of the symptoms of a cerebellar neoplasm, and a bilateral papilledema of 5 diopters There, as in our case, there was a progressive recession of the papilledema

Papilledema occurs chiefly in brain tumors, but it occurs also in hydrocephalus, cerebral abscess, tuberculosis, and syphilitic meningitis, in meningitis serosa, Quincke's edema and, rarely, in infectious meningitis It is seen in cerebral hemorrhage, basilar, particularly late subdural hematoma, pachymeningitis hemorrhagica, sinus thrombosis, marantic and infectious, perhaps in association with a secondary hydrocephalus, cerebral aneurysm, deformities of the skull, such as oxycephalus, rarely in nephritis, arteriosclerosis, chlorosis, polycythemia, leukemia, and scorbutus, in some infectious diseases, as whooping-cough, tetany, lead palsy, in some cases of infectious myelitis, sinus disease, Basedow's disease, multiple sclerosis and encephalitis lethargica

It is obvious that in many of the diseases mentioned what has been reported as papilledema or choked disk probably was a papillitis For example, the cases following sinus disease which produce a peri or interstitial neuritis or a papillitis, cases occurring in infectious diseases, many of the cases of syphilis, multiple sclerosis, lead palsy, myelitis Basedow's disease, nephritis, arteriosclerosis, at times in chlorosis, and so forth The rarity of the occurrence of the latter group in general may be noted from its occurrence in multiple sclerosis when up to 1911 only 9 cases had been recorded

Parenthetically, the procedure of determining the cerebrospinal fluid pressure to differentiate a papilledema with a high pressure from a papillitis with a normal pressure *cannot be condemned too strongly*

In deciding what I should present to you today I felt that a discussion of a few problems dealing with difficult situations would be more profitable than a collective review, and I have therefore taken in alphabetical order the first 100 cases of brain tumor from the service of Dr Loyal Davis, who has generously permitted me to analyze this material A number of these patients were examined by me In addition, I have selected a group of some twenty-one tumor suspects, whose cases offered several problems in diagnosis, ten brain abscesses, and a few other cases

Although papilledema is most frequently found in brain tumors, it is necessary to note that absence of a papilledema does not exclude this diagnosis providing other signs are present The frequency of papilledema varies slightly in its notation in different clinics, dependent upon the material, the care used in verification of tumors, for example, the inclusion of tumor suspects in the group of unverified tumors changes the statistics materially and the disproportion of pituitary adenomata, chiasmal tumors, and so forth, in a small series does the same.

In these 100 cases 84 were verified histologically, and 16 were found, but unverified In the verified group 71 4 per cent showed papilledema In all the cases 73 per cent showed papilledema Excluding 9 cases of pituitary adenoma and two

tumors of the chiasm which, as expected, showed a primary optic atrophy, of the remaining 89 cases, 73, or 82 per cent, showed papilledema

Although it is stated that in a general way the papilledema often is more marked on the side of the tumor, this certainly was not the case, and no conclusion as to lateral diagnosis could be made from these cases

Although parasagittal tumors are said to produce increased intracranial pressure without papilledema, the 1 case in this group showed a papilledema of 3 diopters swelling with enlarged blind spots It has been noted that prefrontal lobe tumors are less frequently associated with papilledema than those of the postfrontal region Considering the temporal lobe tumors, of which there were thirteen verified and two unverified, and the parietal lobe tumors, of which there were seven verified, it was found that of 22 cases, 18 showed papilledema of varying degrees, whereas the frontal lobe tumors, eleven in number, showed papilledema in five, congested disks in two, no change in three, and a primary optic atrophy in one case Pituitary adenomata are known to produce primary optic atrophy, unless the capsule of the tumor has ruptured, and of the 9 cases included here in only one, in which the capsule had ruptured, was a choked disk observed

Infratentorial tumors showed a high percentage of papilledema Of fifteen cerebellar hemisphere tumors only two showed normal disks, and in these cases the tumor was located well away from the brain stem, all patients suffered severely from headache and vomiting In 8 of these cases a secondary papillitis with numerous hemorrhages was seen In general, the more marked the secondary hydrocephalus, the more marked was the papilledema Secondary optic atrophy was seen in 4 cases, emphasizing the great danger in neglecting early operation Only 1 of 8 cases of midline tumors did not show a marked papilledema, and this was the only case which did not have a secondary hydrocephalus A larger number of the neuronomata showed papilledema than would be expected, seven out of ten Probably this occurred because many of these patients with

tumor reported to Dr Davis long after the beginning of tumor growth, after they had in some instances obtained a very large size All of the five midbrain tumors showed secondary hydrocephalus and papilledema, although the one intrapontine tumor showed a papilledema because it had occluded the aqueduct It must be stated that in general these tumors produce focal symptoms long before papilledema occurs

Foster Kennedy called attention to a syndrome in which subfrontal abscesses or tumors caused unilateral loss of sense of smell, with ipsolateral optic atrophy or contralateral choked disk Although originally alluding to frontal lobe lesions in general, Cushing has brilliantly pointed out its frequent and characteristic occurrence in olfactory groove meningiomata Frontal lobe tumors in general have not been found to produce the syndrome, whereas in olfactory groove meningiomata at some time or other it is probably very frequently present. Of 2 cases, one showed only primary optic atrophy on one side, the other an ipsolateral optic atrophy and a contralateral papilledema

The tumor suspect material may be divided into cases presenting symptoms pointing to a cerebral lesion, of which there were 15, 5 cases showing interesting signs pointing to lesions around the chiasm, and three infratentorial lesions Of the first group it is notable that in only 1 case was there a marked papilledema, in three, a beginning papilledema, and in 1 what was interpreted as an optic neuritis

The group of tumor suspects formed the most interesting group of cases of all. Many undoubtedly will develop symptoms or signs leading to a correct diagnosis, some of which will be tumors of the brain Necropsy will undoubtedly reveal the presence of tumor in some

Among such cases are included cerebral vascular disease, cerebral syphilis, meningitis, encephalitis, abscess, labyrinthine disease, aneurysm, nephritic edema, and all of the cases of the so-called "pseudo-tumor" Internal hydrocephalus from causes other than brain tumor constituted a large number of these cases This may be the result of cachexia, nephritis lead,

chlorosis, inflammatory disease, perhaps myxedema, and pregnancy The diagnosis of serous meningitis often masks other conditions, although angioneurotic edema may be complicated by cerebral disturbance imitating tumor Arachnoiditis, frequently the diagnosis in tumor suspects, may be but one of the signs of some other pathologic condition

The differential diagnosis of some vascular lesions and brain tumor is often very difficult A man aged thirty-five developed a slight paraphasia, then a numbness in his right side, then temporary loss of vision, from which he recovered in a few hours He worked through the day, but the next day was unable to write, both because of an agraphia and a numbness in the arm Within two weeks he developed an astereognosis and clumsiness of the right upper and lower extremities Upon examination he showed a weakness of the right side with increased deep reflexes, the above-mentioned signs, marked emotional instability, and irritability were present Ophthalmoscopic examination showed normal disks, the visual fields and blind spots were normal The left parietofrontal cortex was explored and was found to be softer than usual and of a modified color A decompression was left and the patient made a complete recovery

A woman aged fifty suddenly felt dizzy and faint, within a few hours she developed weakness of the left side, and when examined several days later she had a left hemiparesis and at times would have a left homonymous hemianopsia Opthalmoscopic examination showed blurred margins of the disks with engorged veins and exudate along the vessels Operation revealed slightly lessened firmness of the right cerebrum, and pathologic report of an astrocytoma was returned

In the first case we had a slowly progressing lesion, which, despite the normal disks, we felt to be a tumor Nothing was found and the patient recovered Of course, syphilis had been excluded

In the second case there was an onset like an embolism with slight changes in the disk, and although a tumor could not grossly be made out an astrocytoma was present

Somewhat similar are the cases in which a differential diagnosis must be made between uremia and brain tumor A man aged forty seven noted a slight weakness in his face, which was so slight that it was considered to be a peripheral facial palsy Within a week he developed slight headache and somnolence Examination showed very slight weakness of the left arm, more marked weakness in the left side of the face and the paresis was definitely of corticospinal origin The non protein nitrogen of the blood was considerably increased, albumin and casts were present in the urine Ophthalmoscopic examination showed slightly blurred margins, full but not tortuous veins, and capillary striæ about the disk The absence of general symptoms and the presence of symptoms of renal deficiency pointed to the possibility of cerebral changes consequent to a nephritis Sudden death occurred as a result of a hemorrhage into a large glioma

Another man aged fifty, with arteriosclerosis, developed severe headaches. One week later he had a generalized seizure and became semicomatose There seemed to be slight weakness of the left side of the body Ophthalmoscopic examination showed bilateral papillitis The blood chemistry was essentially negative A few hyaline casts and a trace of albumin were found in the urine, the total amount of which for twenty four hours was 1500 c c. A tentative diagnosis of cerebral tumor was made, hypertonic glucose solution was administered and was promptly followed by the disappearance of the stupor and no paralysis was noted when the patient regained consciousness The stupor recurred with more marked paresis, and after a few days again disappeared, leaving only a papillitis, and although the patient passed from our observation, subsequent reports indicate that certainly he did not have a cerebral tumor

Common to most of the cases of tumor suspects is the presence of large lakes of subarachnoid fluid and thickened, opaque leptomeninges The signs and symptoms of some of these cases pointed to the frontal lobe, as in 1 case with jacksonian epilepsy, paresis, and apathy Some point to the cerebellopontine angle, with signs of vestibular nerve involvement corroborating

such a localization Even at operation one may be misled, and once what seemed to be a small tumor of the eighth nerve was removed from a man who presented cerebellar ataxia, deafness, and loss of vestibular function Corroborative vestibular tests, negative blood Wassermann reaction, bilateral papilledema were present He later developed a third nerve palsy and the "tumor" proved to be granulomatous tissue

Another man developed tinnitus, vertigo, headache, loss of hearing, and loss of labyrinthine function on the left side, with cerebellar ataxia No papilledema was found At operation a large arachnoidal cyst was found about the eighth nerve Serologic tests were negative

Another case was that of a woman who suffered from headache, tinnitus, and vertigo for two years, who developed vomiting, unsteadiness of gait, and falling to the left Hearing and labyrinthine tests pointed to left cerebellopontine involvement An ophthalmoscopic examination revealed engorged, tortuous veins, but no elevation of the disks An arachnoidal cyst along the eighth nerve was found

A man suffering from severe headaches, the cause of which had been attributed to an infected sinus which was operated upon, developed further symptoms of vomiting, fainting, diplopia, cerebellar ataxia, and labyrinthine examination showed signs referable to a lesion in the mesencephalon Marked papilledema and papillitis were present Marked internal hydrocephalus was revealed on one of the exceedingly rare times ventriculography was used Upon cerebellar exploration considerable subarachnoid fluid was released from under a thickened leptomeninges, and two years afterward he has no visual disturbance, no headaches, or other symptoms

The temporal lobe may be indicated as in this case, wherein the patient developed uncinate seizures, preceded by an aura of the odor of a skunk He suffered from headaches, showed increased deep reflexes on the left side, had a bilateral papilledema with enlarged blind spots, and a concentrically diminished field of vision in the left eye Upon operation a large amount of cerebrospinal fluid escaped under great tension, the headaches

have disappeared, and vision is normal, but the man still has an occasional uncinate fit.

The following case is of great interest A child developed headaches, one month later drowsiness and blurring of vision occurred, then stupor, pain in the left eye, and complete loss of vision Three weeks later there was pain in the right eye and blurring of vision, finally ending within a few hours in complete blindness On the right side a marked papilledema was present, on the left the disk looked pale At operation the right optic nerve seemed to be about three times normal size, and a large flow of subarachnoid fluid occurred It was thought that the child was suffering from a tumor of the optic chiasm To our great surprise and joy, vision began to return several days after the operation, the fields have progressively enlarged and 20/50 vision returned in both eyes

A man aged forty-five complained of loss of interest, depression, difficulty of concentration, but no retardation He was thought to be suffering from an undifferentiated depression Several weeks later he suddenly developed dimness of vision in the right eye Upon examination there seemed to be slight weakness in the left arm and leg The sense of smell was diminished, the right eye showed a papilledema, the left disk was normal, the blind spot was enlarged on the right A right subtemporal decompression was left after an exploration of the right frontal revealed only a large gush of subarachnoid fluid He returned in one year showing exactly the same degree of papilledema and a pallor of the opposite disk Is this a left olfactory groove meningioma? The anterior horns of the ventricle are normal in size and position

A middle-aged woman partially lost her sense of smell two years before dimness of vision in the right eye was noted A markedly pale disk and central scotoma pointed to a retrobulbar lesion She developed a transitory analgesia of the right ophthalmic division of the fifth nerve, certainly a clear indication of a basilar lesion of the right anterior fossa Operation revealed a large subarachnoid cyst and nothing else

It is notable that the cases with so-called "arachnoiditis"

were associated frequently with changes of the optic nerve Never were we satisfied that the change was that of a real papilledema Circumpapillary striæ, exudate, hemorrhages in the retina were usually present In many of the cases without arachnoiditis, and in a few that were reported by the pathologist to be encephalitis, no changes in the disks were found Very marked changes in the disks with few general or focal signs usually pointed to inflammatory lesions, as in the case of a woman with some headache, diplopia, failing vision, deviation of her eye to the left, and little else She had a marked papillitis with many hemorrhages, and was found to be suffering from cerebral syphilis

In 15 cases of brain abscess only one patient had a normal disk, six had papilledema, and three papillitis It was noted, however, that in addition to the papilledema, papillitis was superimposed in 4 cases

In this connection 2 cases may be mentioned One case was that of a child who, after a few days of fever, headache, and vomiting, slowly developed paresis of the left side, stupor, rigidity of the neck, and other signs of meningitis The slowly progressive and complete hemiplegia led to a diagnosis of abscess, even after the spinal fluid revealed pneumococci and a purulent fluid Operation did not reveal an abscess and the disks were normal The patient made a complete recovery

Another case is that of a man who suddenly developed weakness in his right arm, in a few hours in his leg, then an aphasia, hemianalgesia, homonymous hemianopsia, and stupor His temperature was 102 5° F , and there was a leukocytosis of 15,000 There was no pathology in his ears or sinuses Rigidity of the neck and a Brudzinski sign were present The spinal fluid showed 1800 polymorphonuclear leukocytes to the cubic millimeter The cultures from specimens obtained on three successive days were negative The deep-seated lesion suggested an abscess, which operation failed to reveal The disks were normal The patient died on the fourth day of illness

It seems to me that several things have been pointed out First, the need for a clear definition of papilledema, papillitis

and optic neuritis Second, the development of methods for ophthalmologic differentiation of these conditions Third, the necessity for close co-ordination of ophthalmologist and neurologist, to the end that careful and sustained observation will aid in the differential diagnosis of the existence or abscence of tumor, of the existence of other intracranial diseases, of the localization of the tumors, and of the differentiation of inflammatory brain disease

PRESBYTERIAN HOSPITAL

THE ANEMIAS OF INFANCY

I WANT to discuss for you this morning the question of anemias in infancy So much has been written on this subject that I can scarcely hope to do more than present the unsolved problems and try to summarize the subject in such a way that it will be comprehensible

The old idea of the anemic child was that any pale child was anemic This, of couser, is fundamentally wrong Pallor is a symptom of anemia, but it is not anemia The pallor is due to decrease in the amount of hemoglobin beneath the surface of the skin This decrease may, of course, be due to anemia, but on the other hand, it may be due to a temporary or sustained contraction of the peripheral vessels Again, our ideas of anemia in childhood have changed materially with the accumulation of data regarding the normal in the blood picture It will be seen from the table of Lucas that there is a marked

TABLE*

Age	First twenty four hours.	Third to fifth day	Fourteenth day	Six months.	First year	Second year
Red cells	5,500 000 to 8,000,000	5 000 000 to 8,000,000	4,500,000 to 6 000,000	4,000,000 to 5,000 000	4,500 000 to 5,000,000	4 800,000 to 5,300 000
Hemoglobin	95 to 105	95 to 110	80 to 100	75 to 90	80 to 95	80 to 95
White cells	15 000 to 25 000	12,000 to 18,000	10 000 to 15,000	10,000 to 15,000	9,000 to 14 000	8 000 to 13 000
Platelets	250 000 to 450,000	300 000 to 500,000	250 000 to 450,000	200 000 to 400,000	200 000 to 400 000	200 000 to 350 000

*According to Lucas, "Blood and Blood Building Organs," Appleton Clinical Pediatrics, Vol. 11 p 38.

variation in the normal both in respect to hemoglobin, and red-blood count, and white-blood count in the various ages of infancy and childhood We, therefore, cannot use the adult normals as a gauge of the infant's blood, but must use data on the child But even here the variations are quite marked and we must be careful in our interpretation of the facts presented us in a given case

There is another side to anemia in infancy which I have never seen emphasized How many times have all of us seen at autopsy the body of a poor, marasmic infant, the organs of which were definitely pale, and yet the blood-count before death showed that the hemoglobin and red cells were not only normal, but a definitely high normal Can one say that simply because this child had a high blood-count it did not have anemia? Personally I think not Anemia is not determined by blood-count, but by blood-volume and blood-count in their relations to the body tissue Therefore, it does not seem unlikely that children may in certain instances have a high blood-count and yet be anemic This phase of the question we will not discuss further here except as instances arise which can be explained only on this basis Until we get better clinical methods for determining blood-volume we are not going to be able to really get at the fundamental questions of anemia At the present time we are forced to determine the question of anemia almost altogether on the blood-count, and the following instances which I want to present to you now will be estimated on that basis only I shall not include in this clinic the matter of anemia in the newborn, a chapter as yet almost unwritten, but one which contains much interest to him who is seeking knowledge of anemia The number of cases is as yet too few to be able to draw adequate conclusions In the anemias of infancy, as in those of older children and adult life, we have to consider those forms of anemia which apparently are due to some condition which can readily be recognized These anemias are ordinarily regarded as secondary and spoken of as such The other type of anemia is that which in adult life is spoken of as primary largely because the ultimate cause of this anemia is not apparent

Such anemias are occasionally encountered in infancy and form a peculiar chapter, in that some of them are not simulated by anemia in later life

Of the secondary anemias peculiar to infancy may first be mentioned that type of anemia which we most frequently encounter in the premature infant An example of this is little Martin L (173211) who entered the Presbyterian Hospital on December 10, 1923, at the age of three months The baby at the time of entrance weighed 4 pounds and 12 ounces The history was that he had been born in a seven and one half months' pregnancy and weighed 3 pounds with his clothes on at nine days of age He was delivered with forceps, as the membranes had ruptured two days before without any sign of labor The child had vomited some and had had a cyanotic attack just after birth The child nursed for the first month and had a slight upset which was treated with some pills given by a physician The bowel movements became green and watery (it seems likely that these pills must have been calomel) A second physician diagnosed anemia and put the child on Nestle s food, the child having been taken off the breast during the early part of its original trouble After this the child became constipated for which it was given syrup of figs and enemas A third physician was then called who diagnosed indigestion with colic and put the child on Horlick's malted milk with some medicine The gas was relieved It was kept on this for about a month and gained slightly It was then taken to an Infant Welfare Station where powdered albumen milk was advised, but there was some difficulty about taking this

The physical findings in this child are of no importance from our standpoint The child entered the hospital with a blood count of 42 per cent hemoglobin, 2,250,000 red cells, and 7500 white cells

We see here a rather profound anemia, but, unlike the anemia which is encountered in most premature infants, there is no tendency for this to be of the chlorotic type This child was treated by means of transfusions of whole blood intraperitoneally It left the hospital on February 21st with a weight of 6 pounds,

2 ounces, and a blood-count of 70 per cent hemoglobin, 3,460,000 red cells, and 8200 white cells The feeding while in the hospital consisted of albumen milk with dextri-maltose

Aside from the fact that this is not of the chlorotic type, this child shows the characteristics of a secondary anemia which is so frequently associated with prematurity This anemia does not develop before the second or third month and usually from then on if not properly cared for shows a tendency to get worse rather than better There would seem to be some reason to suppose that the anemia of the premature is a deprivation anemia As you know, the last two months of intrauterine life are those in which the most marked deposit of iron occurs in the organs, especially in the liver and spleen This deposit of iron probably acts as a reservoir upon which the infant may draw during his first year of life If that deposit is prevented or decreased, it would seem that this reservoir would become exhausted earlier and as a result an anemia develops and as so frequently happens, it would seem that this anemia would be of the chlorotic type The treatment of this type of anemia is, of course, comparatively simple The supply of iron to the individual, if properly absorbed, seems to meet the need and results in rather rapid recovery It is very hard, however, in the average case to meet this situation by the use of iron by mouth, whether in the form of drugs or food It would be difficult to say that the failure of such therapy was due to the failure in the absorption of the iron from the gastro-intestinal tract, but the results from blood introduced intraperitoneally certainly suggests that such is the case These children offer the best subjects for intraperitoneal transfusion, and unless some complication arises, such as respiratory infection, etc, usually show very marked improvement on this method of therapy

Another type of secondary anemia, which is often spoken of, is nutritional anemia In my experience pure nutritional anemia has been of rather unusual occurrence When I have stopped to analyze cases of secondary anemia which have occurred in my wards, I have found very few that would stand the test of

searching criticism Nearly every case has had in addition to nutritional disturbances some history of infectious conditions and I, therefore, have found it very difficult to present to you a pure case of nutritional anemia It is well known that in countries where goat's milk is used extensively as a food among children, anemia of a rather severe degree is quite common This anemia is spoken of as goats' milk anemia I, however, have had practically no experience with goat's milk in feeding babies and can, therefore, present you at this time no case of this disorder which has been adequately studied by me There is, however, in the wards at the present time this case of baby Edward M (No 229993) who entered the Presbyterian Hospital on October 24, 1928, at the age of seven weeks The history on entrance was that this child had been having four to eight stools a day since birth These stools have been watery at times, described as being "like paint," sometimes of mushy consistency, and of a mixed greenish yellow color Very rarely have the stools been formed The mother states that she has taken the baby to the Infant Welfare Station since he was four weeks old and has tried to follow the instructions The infant has always taken his feedings well, but has not gained in weight At birth he weighed 5 pounds, 7 ounces, and at the present time weighs 15 pounds, 8 ounces The infant occasionally has a cold The child was born on September 3, 1928 at St Luke's Hospital Its birth weight was 5 pounds, 7 ounces, and it cried immediately No instruments were used and the pregnancy was normal in time Repeated attempts to get evidence that the child was premature have resulted in failure The mother says that unquestionably this baby was a full term baby The child was given breast milk for the first four weeks and then put on a formula of whole milk, water, and sugar The baby entered the hospital with a weight of 5 pounds, 9½ ounces At the present time, at the end of two months and a half, it weighs only 7 pounds, 5 ounces The child's stools have been relatively normal since he came into the hospital and there has been very little vomiting With the exception of the last few days, his temperature has been within normal limits On

January 15th his temperature rose to 102 4° F and on January 17th got as high as 104 6° F

The interesting thing about this child is that on entrance he had a hemoglobin of 95 per cent, red cells 4,330,000, and 13,500 white cells That was on October 24th No blood-count was made on this child again until November 12th At this time the child had shown a gain of approximately ½ pound in weight and was in every way much better The color was not materially changed, if anything the child had a rosier hue and yet at this time the hemoglobin showed 40 per cent, red cells 2,210,000, white cells 13,400 Why the change? It seems to me that this case is one where the volume of blood was reduced following diarrhea For a while apparently (from the blood-count) this child had no anemia That this was not the true state of affairs, but that the blood was concentrated and insufficient in amount for the body needs is, I think, the explanation There is, as I see it, no other way of accounting for this marked change in the blood-picture in the course of less than three weeks and certainly the clinical condition of this child would not bear out the statement that he was suffering from any destructive process which would produce such a lowering of the hemoglobin and red cells There was no hemorrhage at this time There was no infectious process and the general condition of the baby was far better than at the time of entrance This child, it seems to me, represents a form of nutritional anemia which is relatively clear Without any treatment, without change, so far as its general condition was concerned this baby gradually became better so that by January 3d the hemoglobin had increased to 60 per cent, red cells to 3,420,000, with a white-cell count of 9800 What the effect of the acute upper respiratory infection, which developed on January 15th, will have on the blood-picture is uncertain at the present time The present blood-count shows 43 per cent hemoglobin, 3,130,000 red cells, 29,550 white cells, 66 per cent neutrophils, 26 per cent small mononuclears, and 8 per cent large mononuclears

The explanation of nutritional anemia is hardly as simple as that of the anemias in the premature While there may

be something in the fact that the food of the infant contains comparatively little iron, this can scarcely be regarded as the chief factor in the production of nutritional anemia If it were purely a matter of deprivation certainly more children would develop secondary anemias of a nutritional nature than are to be found at the present time If, on the other hand, there develops some toxic substance whose action is destructive to the red cells and hemoglobin, such a substance has as yet not been recognized It seems to me that it is only on the supposition that such a substance is present that we will be able to explain these nutritional anemias If anemias of this type are purely nutritional they will usually react to blood transfusion Many of them, however, show a definite tendency to improve as in this case, without resorting to such therapy provided the nutritional disturbance is properly treated These cases, however, are much more stubborn than those which are encountered in the premature

Another question which may be raised regarding nutritional anemia is why should anemia develop in one child and not in another, although the conditions of feeding and environment may be practically the same At the present time we answer this question clinically by saying that there is a difference in the constitution of the child However, no one has explained as yet the constitutional factors which enter into the production of an anemia We are at sea to explain the individual conditions of this Even in cases of goats' milk anemia only a comparatively small portion of the children taking goats' milk develop anemia There must, therefore, be constitutional factors in this as well as in other types of nutritional anemia

The third type of secondary anemia in infancy and the one which, in my experience, has been far the most frequent is that which is associated with infectious conditions These, of course, are usually infections of the upper respiratory tract, and offer at times difficulty in really arriving at the true cause of anemia since many of these children are in a poor state of nutrition

Rose O (No 226706) entered the Presbyterian Hospital

on July 23, 1928, at the age of two years This child was a Mexican and had been sick for four weeks with definite evidence of respiratory infection, with cough, fever, and vomiting The history was that the child had started in with pneumonia and that the crisis had occurred two weeks previous to admission, but that recovery did not follow and the child still had a cough, fever, and vomiting

On physical examination she had rapid respiration, the respiratory rate being 28, with dilatation of the alæ nasi Vocal fremitus was absent over the left base, back and front, and decreased over the entire left lung Percussion note was dull on the left side below the fourth rib in front and below the fifth in the axilla and at the level of the fifth spine in the back The breath sounds were normal on the right side, but on the left side posteriorly they were absent and over the rest of the left lung were distinct and high-pitched The case then was one of unresolved pneumonia, the diagnosis being confirmed by x-ray

The child recovered and left the hospital on August 16th The only history of any previous infection was that of whooping-cough which had occurred when the child was six months old This child showed a hemoglobin of 42 per cent , with 5,600,000 red cells and 18,150 white cells on entrance This was not materially changed and she left the hospital with 48 per cent hemoglobin, 5,490,000 reds, and 10,300 whites

Here was a mild type of chlorotic anemia in an infant two years old which was evidently the result of an infection consisting of lobar pneumonia followed by unresolved pneumonia

Another case of somewhat more severe type is that of Jacob D (No 199316), who entered Presbyterian Hospital on April 29, 1926 at the age of eight months He came in because he had been vomiting He had had an intermittent cough which had lasted for four months, discharging ears for four months, and two weeks respectively, and loss of weight for two weeks previous to entrance Evidently the child had had a rather severe infection with thrush for two weeks before coming into the hospital At two months of age the child had contracted pneumonia from which he recovered without much difficulty,

though the cough persisted and he had cried a great deal since The child had had profuse night sweats and continued to be in poor health until four months of age when the left ear began to discharge, and at five months the child had another respiratory attack, probably a pneumonia From this attack he had not entirely recovered at the time of entrance Two weeks before entrance both ears began to discharge This child was a full term baby of normal delivery

The thing which struck one on physical examination was the pallor This was very marked There was some discharge from both external auditory canals There were a few crackling râles at both bases posteriorly and a slightly positive d'Espine sign There were no other findings of note

This child ran a rather high temperature, going up to 104° F for the first week in the hospital, and thereafter some temperature for another week, which at one time reached a peak of 104° F After that the temperature was well within normal for ten days, when there was another slight rise of temperature for two or three days, which continued irregularly throughout his course in the hospital It was only in the last week that his temperature stayed regularly below 100 degrees

This child had a most interesting blood count On entrance the hemoglobin was 45 per cent, red blood cells 2,180,000, white count 30600 There was a marked anisocytosis, with microcytes and macrocytes, poikilocytosis, and polychromatia, with stippled cells, 5 normoblasts and 6 megaloblasts, some with double nuclei, were found

By May 1st the blood count was worse 38 per cent. hemoglobin, 2,040,000 red cells, and 36,400 white cells, with 26 megaloblasts and 12 normoblasts On the third the child was given 155 c.c of the father's citrated blood intravenously, by the anterior fontanel On the sixth the hemoglobin was 46 per cent, red cells 2 230,000, and white cells 12,000 There was only 1 normoblast and 8 megaloblasts in the count On the ninth the hemoglobin was increased to 57 per cent, reds to 2,480,000, and whites to 18,000 Eight megaloblasts and 9 normoblasts

were noted On the eleventh another transfusion of 170 c c. of the father's citrated blood was given with marked improvement By the fourteenth the hemoglobin was 79 per cent, red cells 4,200,000, and white cells 14,800 There was only 1 megaloblast present

This increase was maintained until June 4th, when the count was 81 per cent hemoglobin, 4,260,000 red cells, and 13,200 white cells There was still anisocytosis and poikilocytosis, and polychromatia, but nucleated red cells were no longer present Following this, with a change in the infective process, sometimes worse, sometimes better, the blood-count varied considerably At the time of leaving the hospital, July 6, 1926, the child had 85 per cent hemoglobin, 4,410,000 red cells, and 9860 white cells The red cells were normal in appearance and there were no nucleated reds

Here is a case of very severe secondary anemia on an infectious basis which responded fairly well to the use of intravenous transfusion It had many of the characteristics in the blood-picture of a von Jaksch anemia There was lack, however, of enlargement of the spleen and liver which is so characteristic of that condition This child was seen several times afterward and showed no return of his anemia When last seen he was strong, robust, and was in excellent general health

These cases of anemia, secondary to an infectious process, can certainly not be explained on the basis of deprivation They must be due to a more destructive process of a more or less specific nature Any one who has followed the numerous epidemics of upper respiratory infection knows that in some epidemics anemias of a rather severe degree occur while in others they are not to be seen in any large numbers This must mean that there is a difference in the type of infection Until we know more about the infectious nature and cause of respiratory epidemics we cannot go more deeply into the cause of the anemia which is frequently associated with them It seems not at all unlikely that this anemia is due to a toxin, bacterial in nature, which has a destructive action on the hemoglobin and red cells

I want to show you now a little boy of two years (No *232002*)

who is at present in the hospital He entered the hospital on December 24th, coming in primarily because of blood in his urine This had lasted for two days He had had some swelling of the face for four days and of the feet for one day

Ten days before admission the child developed cold, fever, and pain in his right ear The next morning the ear began to discharge and there had been continuous discharge up to the time of entrance The fever went down after the ear began to drain There was a moderately severe cough at the onset of the illness but this and the coryza had improved in the past few days until two days previously, when he began to appear and feel better Four days previously the mother noticed that the child's face had become swollen This had become worse in the last twenty four hours Two days before entrance it was noticed that the urine was red Since then all the urine voided has been bloody There has been apparently no reduction in the quantity passed and no pain has been associated with presence of this blood On entrance the child's feet were found to be swollen This infection was apparently part of the general infection of la grippe which has been going around and had affected various members of the family

The child on entrance showed much swelling of the face, especially of the eyelids, some swelling of the feet, and a rather rapid heart which was, however, not dilated Aside from a convergent squint which was evidently present before the recent illness, there was nothing special on physical examination except the pallor The ears were discharging as mentioned and the throat was red Here we have a perfectly typical picture of an acute infectious process of the upper respiratory tract and ears, complicated by acute hemorrhagic nephritis The explanation of the anemia which accompanied this is, however not so easy

On entrance this child had 40 per cent. hemoglobin, 3,440,000 red cells, and 28,200 white cells On January 7th this hemoglobin had increased to 47 per cent , with red cells 3,995,000, and with white cells 16,150 Up to that time the child had been making steady progress The blood was gradually disappearing from

the urine, there had been no increase in his respiratory infection, and the child's general condition had improved definitely However, on January 13th the child showed an increase in the severity of his respiratory infection with a rise of temperature to 102° F , vomiting, and redness of the right ear-drum On January 14th his hemoglobin was down to 35 per cent , the red cells to 3,350,000, with white cells 11,800

The analysis of this case is of some interest Is this secondary anemia purely on the basis of an infectious process or is it due to the loss of blood through the hemorrhagic nephritis, or are both factors in the production of the anemia? Probably the last is the true explanation But the experience at the time of recrudescence of his infection leads one to think that the infectious process in this instance is one which is of paramount importance in the production of the anemia The marked drop in hemoglobin and red cells with the recurrence of the otitis media, is a strong suggestion that the condition is much more likely due to the destruction of blood through the infectious process than the loss of blood through the hemorrhagic nephritis

A special type of infectious anemia which is rather frequent in infants is that associated with congenital syphilis Two most interesting cases have come to us in the last few months

Alfred W (No 229989) entered the Presbyterian Hospital on October 24, 1928, at the age of five months He is a negro baby with the following history He had had fever for a day, with cough for a few days, and had vomited once the evening before At one month of age the child was taken to the Infant Welfare Station and from there sent to a free dispensary because of a papular eruption on the palms and soles The blood Wassermann at that time was strongly positive so the mother returned for treatment The child was recommended for hospitalization from that clinic Five days before entrance the child began to cough The cough became increasingly worse, since when it has become almost continuous Apparently the night before entrance the child had had fever and had vomited its 10 P M feeding

To make a long story short, the child was suffering from

acute bronchitis with asthmatic type of breathing Aside from the lung findings, the child showed some cervical adenopathy, a palpable spleen, and some pigmentation on the palms, soles, and back, evidently the remains of the former active lesions of syphilis This child ran a temperature for four days thereafter and at only one time did the temperature reach 100° F He left the hospital on December 5th During his stay in the hospital he was given a full course of twelve intraperitoneal injections of neo arsphenamin, 50 mg each His urine on entrance showed many red cells, with 3+ benzidin test This cleared up quite rapidly and the blood disappeared within a week after entrance Albumin thereafter was present at times and occasionally granular and hyalin casts were found On entrance his hemoglobin was 45 per cent , his red cells 3,610,000, and white cells 18,050 During his stay in the hospital without any special treatment for the anemia and with only treatment for his syphilis the blood showed marked improvement, he left the hospital with 65 per cent hemoglobin 5,160,000 red cells, and 10,350 white cells

The problem here was how much of this anemia was secondary to the syphilitic infection and how much due to the respiratory infection Not only that, but was the hemorrhagic nephritis due to the respiratory infection or was it syphilitic in nature? The course in this case does not conclusively prove either condition The marked improvement in the anemia might have been due to the rapid recovery from the respiratory infection The hemorrhagic nephritis, however, from the course which has already been outlined, must be explained in all probability on the basis of the syphilis and not on that of the respiratory infection Two pictures apparently enter into the production of the anemia in this instance, the acute respiratory infection and the syphilitic infection

The second case, however, is one which is equally interesting and almost parallel in nature This was Laura C (No 231632) who entered Presbyterian Hospital on December 11, 1928, at the age of six weeks This child came in because of tenderness of the knees and elbows. The baby was cared for during the

first weeks of life by a visiting nurse After the nurse had stopped her visits the mother noticed that any manipulation of the knees or elbows caused the child to scream with pain No other joints appeared to the mother to be abnormal There were no other complaints About two weeks after she first noticed this pain the mother came to the Central Free Dispensary with the child, since the condition seemed to be worse An *x*-ray was taken and the blood-test of the baby procured The child was a full-term baby, normal delivery The mother had had three children by another husband, all normal On examination this child showed great tenderness of the knee-joints and arms, especially the elbows, a large, palpable spleen, moderate lymphadenopathy with enlargement of the right epitrochlear, and a small umbilical hernia The urinalysis in this instance showed some albumin and blood with an increased number of white cells The *x*-ray showed a marked periostitis in all the bones of both extremities and definite signs of an epiphysitis, very evidently syphilitic in nature This baby on entrance had a hemoglobin of 29 per cent, 2,200,000 red cells, and 22,500 white

Under the syphilitic treatment carried out in much the same way as in the previous case the hemoglobin had gradually increased to 40 per cent on January 8th, with 3,790,000 red cells and 10,400 white The general condition of the child had improved The urine had gradually improved, the blood having disappeared, and the number of white cells reduced to a very few

On January 9th the child had a slight rise of temperature and developed a rather severe upper respiratory infection The result of this was that on the twelfth the child showed a hemoglobin of 36 per cent, red cells 3,540,000

Here was a case in a baby six weeks old, of a very severe variety of syphilis with involvement of all four extremities, Parrot's pseudoparalysis syphilitica, hemorrhagic nephritis, and severe anemia In this instance there can be no question as to what the cause of the anemia was Unquestionably it was secondary to the syphilitic infection The same may be said

also of the nephritis The child showed a distinct improve ment from the former condition, and then had a slight relapse in the blood-picture due to the presence of the acute upper respira tory infection

These 2 cases showed very definitely a secondary anemia, which is so frequently encountered in cases of congenital syphilis It should be mentioned here that the anemia is not always of this simple variety The clinical picture may in many instances simulate that of a von Jaksch anemia, since the enlargement of the spleen and liver is part of the picture of syphilis as well as of von Jaksch's anemia and the blood picture does not, in some instances, differ materially from that of von Jaksch's in that immature cells including the nucleated red cells, are com monly found This type of anemia secondary to syphilis is, however, not common A typical secondary anemia is found, but not often to the same degree as pictured in these 2 cases

An interesting case which I wish to present to you is that of a negro boy nineteen months old, who entered the hospital on August 27th last, and has been there ever since At the time of entrance the grandmother gave the following history The second week in June the boy vomited after his breakfast two or three times Since then he has been vomiting occasionally after breakfast, but never after any other meal This has been more and more frequent so that for the past two or three weeks he has vomited two or three mornings in succession, then missed two or three days and started again The appetite has remained excellent and he was always ready to eat almost immediately after vomiting The grandmother says he has lost weight dur ing this period, she does not know how much During the early part of this month while under treatment advised at Mercy Hospital, he gained 1 pound and 12 ounces in ten days He picks up between the periods of vomiting, but loses weight again when the vomiting begins On August 22d and 23d he had nine convulsions within twenty four hours These were generalized and each was followed by a deep sleep One is said to have lasted twenty minutes There have been no other con-vulsions There has been no diarrhea or constipation His

stools have contained mucus, but no blood They have been either yellow or dark brown, but never green The pregnancy and birth were normal, though the mother had measles at the time of the child's birth The child was breast fed for five months, after that with condensed milk, and then a cows'-milk formula Cereals and vegetables were added later Cod-liver oil has been given since two months of age, but orange juice only irregularly The mother who was only sixteen, was suffering from tuberculosis in a hospital The father deserted the family and nothing more was known of him

On entrance there appeared a well-developed negro child who did not appear ill He had a few posterior cervical lymph-glands and epitrochlears the size of a split pea There was a peculiar discoloration of the scleræ which consisted of mottled deep bluish-gray color in small patches No other eye findings were noted The mucous membranes were pale The heart showed a slight systolic murmur, but was otherwise normal, as were the lungs The abdomen was distended, the liver and spleen were not palpable There were some signs of healed rickets, such as a rosary and rachitic girdle Otherwise nothing abnormal was noted

During his stay in the hospital a slight enlargement of the spleen was developed which has continued up to the present time Otherwise the abnormal findings were essentially the same as those noted on entrance He is very lively, active, and in a fairly good state of nutrition, as you see At twenty-three months he weighs 22½ pounds Aside from upper respiratory infections he has had very little in the way of illness since his entrance to the hospital At times he seems to have been suffering with abdominal pain, but this does not last long During his stay in the hospital he has usually had a slight fever ranging between 99 and 100° F, even when he has no evidence of respiratory infection

A most interesting fact which has not come out in the history and which was only gained after his entrance to the hospital was that this boy was very fond of paint Soon after coming into the hospital he found ways of chipping the paint

off his bed and eating it, so much so that we had to wrap the entire frame of the bed with bandages Whenever he gets a toy with paint on it he immediately puts it in his mouth and chews the paint off

The blood findings in this case are of peculiar interest On entrance his hemoglobin was 32 per cent , red blood cells 2,850,000, white cells 27,250, neutrophils 36 per cent , small mononuclears 56 per cent., large mononuclears, 6 per cent It is interesting to note that basophils and stippling of the red cells were evident on first examination At that time twenty four normoblasts were noted to 100 white cells This situation has continued almost without change during his five-months' stay in the hospital He has occasionally gone as high as 40 or 42 per cent hemoglobin immediately following transfusion, but has almost immediately dropped back On January 17th it is interesting to note that his blood-count was almost exactly the same as on his day of entrance, that is, 32 per cent hemoglobin, 3,110,000 red cells, and 19,200 whites, 23 per cent neutrophils, 68 per cent. small mononuclears, 3 large mononuclears, and 6 eosinophils To each 100 whites there were 8 normoblasts and 1 or 2 stippled reds There were also noted nuclear inclusions in many of the red cells During the entire stay in the hospital it has been suspected that among other things this might be a case of sickle-cell anemia and repeated attempts to find sickle cells have been made, this with varying success Strange to say that the last examination was the most successful, and in the blood which has stood for a short time there were many sickle cells The interesting thing about this case is that the sickle cells should have escaped observation in spite of diligent search for so long, that is, the number present was not sufficient upon which to make a diagnosis until almost five months after his entry into the hospital The other facts of this picture fitted very well with a diagnosis of sickle-cell anemia, but there are points which are of interest. The first of these is, as to whether the nucleated red cells in such large quantities are to be regarded as in the picture of sickle-cell anemia, and is the large number of stippled reds to be regarded as from the same source, in other words,

is this a case of pure sickle-cell anemia or is it a sickle-cell anemia complicated by some other form?

The symptoms and the finding of stippled cells in the blood are strongly suggestive of lead-poisoning, which might easily exist following the ingestion of large quantities of paint such as this child indulged in For a long while it was thought that this case was one of lead-poisoning with severe anemia, but it seems now that it probably is a case of sickle-cell anemia with perhaps lead-poisoning as a complicating factor The presence of such large numbers of nucleated red cells means, of course, that there must be a great deal of stimulation of bone-marrow

So far we have talked of conditions which for the most part show in the history or in the physical findings some factors which at least in part give an explanation of the presence of anemia We come now to another clinical condition, the nature of which is much more obscure We have seen that in many of the more severe types of anemia in infancy, nucleated red cells and even megaloblasts are sometimes present We have also seen that this immaturity in red cells may also be found in the whites We come now to a condition in which with great regularity both are present Let me briefly tell you of a case which has recently come under my observation

This baby was first seen on September 8, 1928 when he was four months old He was a full-term baby, instrumental delivery, birth weight 7 pounds, 4 ounces He was given breast and supplementary feedings for two weeks, then artificial feeding with whole milk and Karo syrup, and the addition of casec for the rest of the time He had had some sort of gastro-intestinal disturbance of a decidedly minor nature Aside from that he was perfectly well to all intents and purposes The weight when first seen was 12 pounds, $3\frac{1}{4}$ ounces, almost exactly 5 pounds over his birth weight The father and mother were perfectly well and healthy, and this was the only child He had been watched by another physician who had noted early in August that his spleen was enlarged and that he had palpable lymph-glands in the axilla and neck

On the first examination he had 27,300 white cells On

August 14th the spleen was definitely larger and the white count had risen to 67,300 On the twenty first this was 46,600 The blood Wassermann on the child and the parents was negative On September 8th the blood-count was as follows Hemoglobin 73 per cent , red blood cells 4,160,000, white cells 39,350 It is interesting to note that myelocytes were present in the count and also basophils On the first examination when he presented himself to us he had an enormous hard spleen which reached to 1 inch below the umbilicus The liver likewise was enlarged, but not to the same degree, showing about two fingerbreadths below the costal margin There was marked general lymphadenopathy

The only essential change that was made in his diet was to add a small amount of spinach water On September 10th his count showed 60 per cent hemoglobin, 3,800,000 red cells, and 35,200 white cells In the smear 3 megaloblasts were noted It was also noted that a large percentage of the white cells were immature and many could not be classified This has continued up to the present time At present he is an apparently strong healthy baby He weighs about 18 pounds On January 17th his blood-count was as follows Hemoglobin 80 per cent , red cells 4,300,000, white cells 15,000, polymorphonuclears 44 per cent , neutrophils 2½ per cent , eosinophils 1 per cent , basophils 5 per cent , myelocytes 5 per cent , neutrophilic myelocytes 5 per cent , eosinophilic myelocytes 0 5 per cent, basophilic myelocytes 1 per cent., small lymphocytes 30 per cent., large lymphocytes 44 per cent , large mononuclears and transitionals 9 per cent. There was also a slight variation in the size and shape of the nucleated reds Platelets appeared to be about 500,000 The spleen and liver at this time were not materially larger in size, but were apparently not as firm as they were on the previous examination To look at the child you would say that you had a perfectly normal healthy baby He has gone through an acute attack of upper respiratory infection without materially affecting his progress What the future holds for this baby one cannot tell, but if we may judge by his course in the last four months we may expect a gradual improvement This child has all the

earmarks of a von Jaksch anemia pseudoleukæmica infantum The child has shown this progress without any therapeutic measures being employed It is hard to see how the use of a small amount of spinach would produce a result such as this Some children are apparently much improved by the use of blood tranfusions whether it is given intravenously or intraperitoneally

Another typical case of this was in a little girl nine months of age (No 196192) who came to the hospital on January 18, 1926, with a blood-picture of 41 per cent hemoglobin, 2,100,000 red cells, and 3800 to 4300 white cells This child had many nucleated reds and had exactly the same tendency to immaturity in the white cells She stayed in the hospital until March 11th of the same year, during which time she had, on January 26th, 165 c c of her father's blood intraperitoneally She left the hospital in good condition with the blood showing 80 per cent hemoglobin, 3,780,000 red cells, and 3300 whites While there was some aniso- and poikilo-cytosis, no nucleated reds were noted and there were no immature white cells seen She was seen afterward and made an uneventful recovery

That transfusion is by no means always successful is shown definitely by another case of a little girl of two years (No 192746), who entered the hospital on September 18, 1925 Numerous transfusions were given to this child whose blood on entrance showed 51 per cent hemoglobin, 3,810,000 red cells, and 28,000 white This child also showed many nucleated reds and immature cells She left the hospital on October 16th with a blood-count of 83 per cent hemoglobin, 3,910,000 reds, and 3300 whites Megaloblasts were still present and immature white cells She had an enormous spleen and liver which persisted during this entire time

She returned to the hospital on November 4th very evidently worse Her spleen was so large that it pressed on the pelvic bones and caused vomiting by pressure on the stomach This vomiting was relieved by reducing the size of the spleen by means of x-ray treatments On November 4th, almost exactly three weeks after her departure from the hospital, the

child showed 47 per cent hemoglobin, 2,380,000 red cells, and 138,000 white. All the different changes both of red and white cells were noted From that time on she went steadily downhill in her general condition The last blood count was on November 23d and showed 33 per cent hemoglobin, 1,730,000 red cells, and 11,300 white, this in spite of repeated transfusions Removal of the spleen had failed to produce any relief The child died on November 25th No general autopsy was held, but examination of the spleen failed to show any evidence of leukemia

Here is a child who was given every opportunity to react to blood transfusion and yet while there was apparent relief at the start, the condition was not relieved at all in the last few weeks of life Between this case and the one previously mentioned, there is the difference that at no time in the last case had the child failed to show nucleated red cells and immature white cells What this condition is no one seems to know Whether it is a combination of several diseases or whether the whole picture presented represents only variation in the same disease is still doubtful In my experience it has seemed that the younger the child the better the chance of recovery I have never seen this condition in a child over five years This child died as did most of the children over two years

We see, therefore, that the anemias of infancy are varied both as to etiology and as to blood picture In some it is impossible to determine clearly what the causal factor is In others the causal factor is as yet unknown No single form of therapy is to be depended upon in all cases Where possible the etiologic factor should be treated by removing it This is best exemplified in those cases due to congenital syphilis Blood transfusion is in many cases a distinct help In some it seems to be the means of bringing the blood picture back to normal In others of apparently the same type and from the same cause it is ineffective We must, therefore, in treating the cases of anemia in infancy be very careful in differentiating the various causes and institute treatment for the anemia itself, according to the needs in the individual case In my experience iron, when

given in the form of food, is just as effective as iron when given in some other form The best exception to this is to be seen in the results obtained from transfusion In cases of this type it seems to make little difference in the ultimate results whether the blood is given intravenously or intraperitoneally, and since the latter method offers so much less difficulty in technic it is to be preferred

Up to the present time we have used liver or liver extract in only 1 or 2 cases and in these we could not say that any results were obtained Whether or not liver extract is of value in anything other than pernicious anemia is, I think, at the present time unproved

CLINIC OF DR CHARLES SPENCER WILLIAMSON

Research and Educational Hospital

A GROUP OF MEDIASTINAL CASES

Case I.—The first patient I wish to present to you today is a man forty years of age a core maker by occupation who states that he was perfectly well up to four months before his admission He is a Bohemian and of not overly high grade intelligence, but through one of the nurses who has acted as interpreter we think we have a substantially accurate history

Four weeks ago he was awakened from sleep by the spitting of blood and simultaneously was conscious of a severe pain in the left chest He spat up about 2 spoonfuls but since that time has not noticed any more During the first night he fainted three times and his family had the impression that he was very ill Early in the morning they called a physician who gave him some medicine after which he felt much better He remained in bed a week after this because of the pain in the left chest which was so severe that he could not use his left arm. At this time he had no fever and did not cough much and such cough as he did have was unproductive. The doctor he says, gave him some medicine to make him expectorate but this does not seem to have produced the desired results. Since this time he has been up and down and has felt very weak and has had considerable pain but hardly any cough He states that one year ago he weighed 177 and on admission his weight was 154 and he thinks he has lost most of this if not all in the last two or three months.

General and Negative.—The gastro-intestinal symptoms are substantially negative except for some eructations and constipation His respiratory symptoms have been fairly well covered. Since his admission to the hospital his cough has been very scanty and of a mucous character At no time has blood been observed The thoracic pain still persists and when asked where it hurts him he points to the anterior chest region below the left clavicle and extending out into the left arm. When asked he states that he is very short of breath and is becoming increasingly so

He complains of no genito-urinary or nervous symptoms unless the fainting attacks after his hemoptysis are to be construed as nervous His habits are rather correct for a man in his stratum of society in that he neither drinks nor smokes and is very moderate in his use of tea and coffee

Past History.—Aside from the usual diseases of childhood he has had very few infections and says he has had none since arriving at manhood He denies venereal infection He has been married thirteen years has four

children living and well, one child died at the age of one year of uncertain cause

The family history is unimportant

Physical Examination—You see before you a reasonably well-developed but indifferently nourished man of approximately fifty, who has been up and about, although not very spry

Regional Examination—The head is absolutely negative Eyes present no abnormalities and in particular, no edema or inequality of the pupil Neck shows nothing unusual, no adenopathy, no thyroid enlargement

Chest is well developed, no bony deformities and no depressions When we come to examine the lungs we find some rather unusual conditions On the right side both in the front and back we have substantially normal findings On the left side just outside of the nipple line anteriorly we find an area of marked dulness which extends around on the side so that it is almost flat in the axilla Over this area the breath sounds are audible but definitely decreased and have a slight bronchial quality A few moist râles are heard over this area

Heart—Apex beat is palpable in the fifth intercostal space just inside the nipple line, and is approximately of normal intensity and extensity The tones are a little distant but regular in rhythm, with no adventitious sounds

Abdomen—This may be disposed of by saying that it is negative in all respects

Extremities—The extremities show no abnormalities

Reflexes—The usual deep and superficial reflexes are all present and normal

The history, as I have just given to you, is that written by the Junior Intern on the patient's admission, and it might be of interest to note the impression which he had of the case He considered it a pleurisy with effusion So impressed was he with the correctness of this diagnosis that he made an aspiration of the thorax and obtained to his surprise about 15 c c of blood which coagulated immediately He stated that he could have obtained a great deal more and that the fluid obtained seemed to be pure blood since it coagulated practically immediately

I saw the case for the first time four days later and was able to confirm in a general way the physical examination as it has been read to you However, I made the following note

"Close inspection in a good light shows a definite pulsation in the region of the second, third, and fourth rib anteriorly in the left nipple line On palpation the same thing can be elicited" A more careful inspection of the patient in a good light showed that the external jugular on the right side was distinctly larger than on the left, but otherwise nothing new was elicited On finding this pulsation I need hardly say that our suspicions of aneurysm were at once aroused and we took the patient immediately to the fluoroscopic room where a rather unusual state of affairs was revealed To begin with, the heart was pushed over toward the right to a degree that was not at all suspected in the physical examination The dulness which we had found in the first three interspaces and extending out to the nipple line was caused by a large mass casting a very dense shadow, as you will see in the *x*-ray in the

shadow box (Fig 37) This mass is quite distinct from a diffuse shadow in the lower part of the left axilla which is probably due to either a little fluid

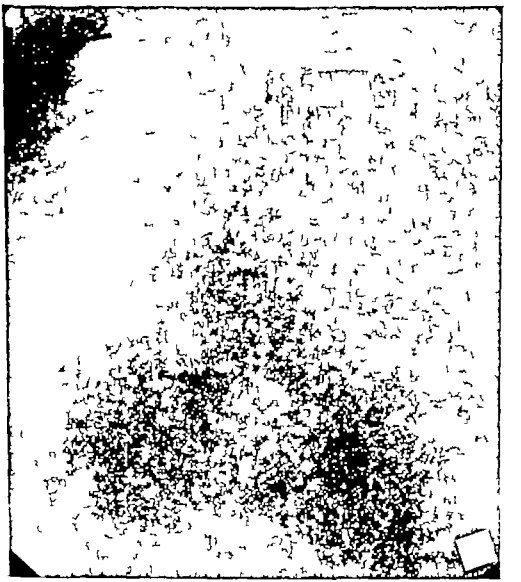

Fig 37 —Large mass on left side. Note the smooth even upper margin

or a thickened pleura The Roentgenologic Department was inclined to think that the pulsation in the mass which could be definitely made out was probably a communicated and not an expansile pulsation

Now that is about where the case stands at the present moment We may dismiss the idea of free fluid in the left chest as being responsible for all the symptoms On the other hand the diagnosis is by no means so clear as regards this mass itself When the aspiration of the thorax was made by my intern, what did he get into? Either the fluid, if there be any in the chest, is hemorrhagic or what is equally likely since he went in quite

far with a long needle, he may have gone into the mass itself. I am rather inclined to the latter hypothesis The *x*-ray diagnosis of the case was a probable tumor of the left lung, with a small amount of fluid in the left pleural cavity Several more *x*-rays since then brought back the same report Personally I regard the case as one of the most interesting from a diagnostic standpoint that we have had on the floor for a long time I think I can demonstrate to your entire satisfaction that there is a definite pulsation just below the left clavicle to the left of the sternum, while slight, it has been found every time it has been carefully looked for On the other hand, we have never been able to elicit a tracheal tug It should, however, be remembered that even with an undoubted aneurysm a number of conditions have to be fulfilled before a tracheal tug can be elicited The pulsation under the fluoroscope is said not to be expansile, but rather a mere rising and falling

Now let us consider the most probable explanation of these physical findings First, let us consider the roentgenologic diagnosis of a probable lung tumor This could hardly be anything else but a malignant tumor, since, as you will remember, it started with the expectoration of blood and the patient has been losing weight steadily ever since To my mind, however, there is a very serious objection to this, namely, that in the time he has been in the hospital, and according to his statement for a long time before, he has had comparatively little cough and only a little mucous sputum This is in striking contrast with what happens in proved cases of malignancy of the lung, where we have almost invariably a persistent cough with a thick, tough blood-stained sputum, often looking like currant-jelly It is almost incredible that a primary malignant tumor of the lung that has once started to bleed should then cease to bleed for a long time and cease to produce cough and expectoration In other words, while the physical findings might well enough be those of a carcinoma of the lung, the history does not fit into this diagnosis

Secondly, we might consider the possibility of a localized and encapsulated accumulation of fluid With this possibility

in view, we asked the surgical department to see the patient with us and I think at first they rather leaned toward this explanation They made a paracentesis in the anterior axillary line two intercostal spaces above the point where my intern aspirated blood, and again obtained 15 c c of blood which clotted at once This, of course, made an ordinary accumulation of fluid highly improbable and an accumulation of blood would

Fig 38 —Note how the heart is pushed way over to the right by the increasing size of the mass

seem still more improbable unless a very vascular tumor or an aneurysmal sac had been tapped by the needle I do not believe that I stated that the sputum was examined many times for tubercle bacilli with negative results and that the aspirated fluid showed nothing out of the ordinary Now it seems to me that in spite of the fact that the tumor showed no expansile pulsation under the fluoroscope, the most probable diagnosis

is aortic aneurysm There are, however, some objections of a very definite nature to this diagnosis The man gives no history of syphilis and his Wassermann is negative Yet we had a patient in the house a short time ago with a frankly negative Wassermann on many occasions who had an extensive luetic aortitis with an aneurysm of the descending aorta This, therefore, is not an insuperable objection It is rather interesting to note that a blood Wassermann on his wife is positive A spinal puncture was done on the patient himself with a negative result

Three Weeks Later—The progress of the case has not been great The pulsation is a little more marked He has lost no more weight, his cough is very slight The pain is as great or greater, and the roentgenologic picture just about the same It is interesting to note, however, that our roentgenologist is now a little less certain of the pulsation not being expansile In the film before you (Fig 38) as in the first one I would like to point out the extremely smooth edge of the mass, a point which to my mind is much more strongly suggestive of aneurysm than of tumor

My main point in presenting this case to you was to call your attention to two things First, how easy it is to overlook the pulsation of the chest unless you get the patient in a good light, calmly sit down and take a careful look at the patient in expiration The usual casual glance will certainly fail to detect a pulsation such as this There is one point which is worthy of notice and that is that aneurysm is at least nine or ten times as frequent as tumor but, of course, this is only a relative point, since one may not exclude a given diagnosis merely because it is rare I think it is not improbable that before long we will have an opportunity to verify or disprove the diagnosis

Case II.—The second patient is a male, forty years of age, colored, who has spent his life in manual labor He entered the hospital complaining of intense pain in the chest, radiating out both to the right and left In addition to this is becoming increasingly short of breath and has noticed a peculiar "whistling sound" to his voice Just recently he says that food seems to stick in his throat He can swallow large morsels, such as meat, but he has to wash them down with water At times he has a little cough but this has not been pronounced

Onset and Course—The patient states that he was entirely well except for some slight hoarseness up to five months ago when he experienced a sharp pain in the lower chest especially in the region of the precordium and the right shoulder The pain was more marked at night and kept the patient from going to sleep or awakened him after he had been asleep During the last two months the sharp lancinating pain has given way to a steady dull pain in the precordium and both shoulders At the same time he noticed that he was short of breath if he did much work. He could not run up a flight of stairs and after walking up two flights of stairs he would have to go to a window to get his breath He states that he has been somewhat hoarse for some years but very definitely so about the time these other symptoms began He has already described the difficulty in swallowing and says this has not made any particular progress in the last few months

About two months after the onset of the above symptoms he developed a slight cough which however has not been constant but during the last few days he has been coughing and expectorating a good deal Patient has been treating himself with home remedies and until coming to the Dispensary the week before he entered the hospital had not been under medical care.

A brief summary of his past history shows that he had the ordinary medical diseases of childhood with pneumonia at twenty four His family history is unimportant. He is married the father of one child by his present wife and one child by his first marriage who died shortly after birth

He had gonorrhea at twenty four a hard chancre at about twenty-one He has been a hard drinker of whiskey up to about a year ago often drinking 2 pints or more a week.

Gastro-Intestinal symptoms are practically negative In the cardio vascular system we have already referred to his dyspnea cough and pain. He has never had any hemoptysis or edema. Respiratory and genito-urinary systems show no unusual symptoms. The special senses are approximately normal

The physical examination made on entrance showed a well developed moderately well nourished colored male weighing 156 pounds as against his normal wight of 180 Temperature 98 F pulse 80 and respirations 20

Head and neck present no abnormalities. Pupils react to light and accommodation

Chest is well developed rather bony Even a superficial examination showed a visible pulsation on the left side in the second costal space and on sitting up this extends over a larger area and is very definitely heaving On percussion we find equal resonance over both lungs except that there is an area of dulness on the left side at the level of the second and third interspace which extends to the midline and to a point 4 or 5 cm to the left. Breath sounds are vesicular

Heart—Apex beat visible and palpable in the sixth interspace and about the anterior axillary line No thrills Heart boundaries substantially normal and the tones pure except that over the aortic cartilage the second sound is distinctly ringing The pulsation in the second interspace has already been referred to

This patient is in many respects very similar to the previous one except that the pulsation is so very definite in the upper part of the sternum and to the left, that one would have to be dull indeed not to think of aneurysm as the most likely diagnosis In addition to this, we have a frankly positive Wassermann and an equally positive Kahn, to say nothing of the fact that the patient tells us he had a chancre which was never treated When we stop to think of the diagnosis of thoracic aneurysm it is quite evident that there are three requirements that should be fulfilled First, we must demonstrate the tumor Second, this tumor must be in a position which makes it possible to be derived from a blood-vessel, and third, it must have an expansile pulsation In this case we have a definite dulness in a position which could hardly be due to a fluid exudate, and which is not due to some consolidation of the lung It is very easy to show that the tumor is pulsating, although the expansile nature of the pulsation is not easy to determine, and the location is such as to make it a matter of great ease for this tumor to be in association with the aorta

It is often quite easy to elicit certain phenomena which point indirectly to the existence of a tumor mass of some sort In other words, there may be pressure symptoms For example, a very common one is a difference in the pulse of the two wrists as a result of pressure or traction by the aneurysmal sac, there may be a difference in the pupils or there may be a difference in the vocal cords, producing hoarseness of voice, and the so-called "brassy cough" The superficial veins of the neck and chest may be engorged on account of the pressure Personally, I do not feel that too much stress can be laid upon these symptoms since, after all, they occur just as readily with a solid tumor as with an aneurysm In other words, they are useful in giving a clue to a tumor, but not to the nature of it Again, I want to lay stress upon the necessity for looking for a pulsation which in this case is easy to see If one stops to reflect on the course of the thoracic aorta and how, starting from its deep origin at the aortic orifice it comes forward to the chest wall in the second right interspace, then goes obliquely across and backward to

extend along the left of the spine, it is quite easy to understand that the symptoms of pressure would depend upon the situation of the aneurysm Here again, only general statements are admissible Most aneurysms of the first part of the aorta and the beginning of the arch present to the right of the sternum and are readily made out by physical examination Hence, the oft repeated designation of "aneurysm of physical signs," as applied

Fig 39 —Aneurysm of the arch and descending aorta

to them. Aneurysms of the descending aorta present behind and to the left of the sternum, erode the vertebral column early and produce pulsations in the back On the other hand, the aneurysms of the transverse part of the arch often grown downward and cannot be made out, at least when they are small, by even the most painstaking physical examination They do, however, produce certain pretty well defined symptoms, such as

a hoarse voice, a brassy cough, laryngeal paralysis, and in most cases a well-defined tracheal tug This is the type of aneurysm that so often is revealed at autopsy and totally unsuspected during life It is in this class of cases particularly that the *x*-ray has been a godsend to us I recently saw a case with a large aneurysm which had been regarded as a tuberculosis and sent west with hopes of recovery Indeed it was seeing this case which suggested the desirability of showing these cases

The *x*-ray in this second case (Fig 39) shows what we already had reason to think, namely, an involvement of the arch and a considerable portion of the descending aorta This latter part could hardly have been recognized in any other way

There seems to be a general impression abroad that aneurysm is a rather uncommon disease and I know that in some countries, especially Germany and Austria, it is relatively uncommon as compared with England and the United States I do not know whether the statistics in the last few years bear out this old assumption I do know in the last few years, in Chicago at least, aneurysm is a very frequent disease There are four in the wards just at the present time and another one died last week I may say parenthetically the one dying last week showed a very small aneurysm of the lower part of the descending aorta which had eroded the spine and which in spite of repeated *x*-ray examinations had not been discovered, probably because of the fact that a large carcinoma which was the cause of death led us to infer that the pain was due to the carcinoma, whereas, as a matter of fact, it may well have been due to the erosion of the vertebral column

Case III.—I have another patient whom I want to present briefly to you because he again shows a variant of the mediastinal picture

He is a white male, fifty-eight years old, by occupation a laborer, of American nativity His complaints are first, dyspnea, second, precordial pain radiating down both arms to the elbows, third, loss of strength, fourth, edema of the legs and fifth, hemoptysis on two occasions

Onset and Course.—Four years ago he developed a severe precordial pain paroxysmal in nature and worse at night He is a big burly fellow but he says the pain is so bad that he can hardly stand it He has had some pain every day since the onset four years ago During the past three years he has

become very dyspneic on the slightest exertion and lately he says that when the precordial pain becomes very severe, he becomes choked up and is unable to get his breath During the past two years his legs and feet have become markedly edematous on many occasions the edema extending from the feet up to the knees During the week before his entry the precordial pain has radiated down both arms to the elbow and is especially pronounced in both axillæ. About eight days ago on two successive days he had a brisk hemoptysis losing about a half glassful of practically pure blood each time The pain and dyspnea have both been increasing markedly in intensity during the last three weeks

General and Negative —Gastro-intestinal symptoms are negative. Of the cardiovascular symptoms the edema and dyspnea, precordial pain have already been referred to

Respiratory —He says he has occasional attacks of coughing no night sweats

Genito-urinary and nervous symptoms are absent. Special senses show no abnormalities

His past history shows nothing of consequence but typhoid at thirty two followed by pneumonia He denies lues but his blood Wassermann shows a four plus positive as does also the Kahn

The physical examination on entrance showed nothing especially abnormal with the head and neck.

Chest —Slightly emphysematous expansion good equal on two sides A careful inspection showed on admission as well as now a marked pulsation both visible and palpable strongly heaving in character in the second right interspace The area of pulsation is about as big as the palm of one's hand and involves the upper part of the sternum as well The lungs present no abnormalities. The heart has its apex beat in the sixth rib in the anterior axillary line. The right heart boundary is distinctly farther out than normal fully 1 or 1½ inches at the level of the fifth rib Over the area of the pulsation there is a marked dulness A loud systolic murmur is heard over the entire precordium but especially loud in the second right interspace

Abdomen —The liver edge is about three fingerbreadths below its normal position and very tender to pressure. The spleen is not palpable.

The extremities show a well-defined edema extending up to the knees.

The deep reflexes are all normal The urine and blood present no abnormalities The blood pressure in the right arm is 160/94 and in the left 180/100

Now let us consider what we have before us I think you will agree with me that there are two distinct conditions One is a very evident myocardial insufficiency, as evidenced by the swollen and tender liver and the edema of the feet. I do not wish to enter into the differential diagnosis of this too carefully or to discuss how much of this may be due to the rather moderate degree of hypertension I believe I neglected to say that

the pulse is absolutely irregular, with a typical picture of auricular fibrillation So I think we may say with a high degree of probability that a chronic myocardial degeneration aided by a moderate degree of hypertension is responsible for the cardiac decompensation

Fig 40 —Aneurysm of the ascending aorta and the arch (Note on going through press Five months after this picture was taken the pulsation had extended up to the right clavicle Innominate involvement?)

The second component in the diagnosis relates, of course, to the pulsation over the upper sternum and to the right In a man of fifty-eight years with intense pain in the upper chest, with a four-plus Wassermann, expectorating blood from time to time, and presenting a very definite pulsation, the diagnosis of aneurysm can be made with almost perfect certainty without the aid of the x-ray This, however, has been made and

shows a very evident aneurysm (Fig 40), involving not only a considerable portion of the ascending aorta, but a considerable portion of the arch as well I may say that this is what usually happens The clinician makes up his mind about how extensive the aneurysm is and then the roentgenologist tells him very clearly that it is much more extensive than he had supposed At least, I am qute free to say that that has been my personal experience One symptom is a little difficult to explain, namely, the inequality of the two pulses The x ray does not disclose the reasons for this but I imagine it is traction on the right subclavian in some way or other, or perhaps an involvement of the orifice of the subclavian by an especially marked aortitis On looking for the very much talked about tracheal tug we find that this patient does not show it Indeed, my own experience with the tracheal tug is, that while frequently present it is just about as frequently absent and that, too, in the very cases where one would suppose it would be most likely to exist. The particular point to be stressed in this case seems to me to be that, since aneurysm is due to aortitis and since it occurs at an age and under conditions when chronic myocarditis is very prevalent, it may be often masked to some degree by the existence of a myocardial insufficiency From what the patient tells me, I suspect that his physicians have regarded his case as one of myocardial insufficiency only and have looked upon the pain in his chest as angina pectoris

Case IV —The last patient is a colored male fifty five years old who has been a cook all his life. His entrance complaints are loss of weight amounting to 76 pounds in two years Along with this he has had a good deal of headache and in the last month has developed marked dyspnea and a cough at night. During the last week or two he has had severe precordial pain His intelligence is not of a very high grade and the history is more or less unreliable because of this fact The point which has impressed him most and about which he seems most certain is the 76 pounds loss of weight of which 20 have been lost in the past two months.

General and Negative —Aside from some constipation gastro-intestinal symptoms are absent In the cardiovascular system the dyspnea and precordial pain have already been referred to Edema is absent

Respiratory —He has had no cough or expectoration no night sweats or fever

Genito-urinary and nervous symptoms are absent and the special senses are good

Past History—Aside from the usual diseases of childhood, he has had nothing but the shingles in 1917 He had a severe accident in 1907 but broke no bones

His family history is negative

He has been married twice, the first time twenty-seven years, with no children but two miscarriages at the fifth month, second marriage one year, no children, no miscarriages He denies lues but he shows a four plus blood Wassermann and also a four plus Kahn

Physical examination shows a big, well-developed negro male in very poor nourishment, weighing now 159 pounds in contrast to the normal of 235 pounds

Head and neck are normal except that in the episternal notch a very distinct pulsation can be felt This symptom is exceedingly valuable if one can be sure that it is not the carotids which one is palpating The pulsation is characteristic and I feel sure in this case that it is the aortic arch itself

Chest—On inspection a very distinct pulsation is seen on the right side in the first intercostal space and above the clavicle, extending from about the outer end of the inner third of that bone under the sternocleidomastoid and is continuous with the pulsation already referred to, as being felt in the episternal notch This pulsation is strongly heaving and can be felt as it comes out above the clavicle to be distinctly expansile in type The general impression created by the pulsation is one of tremendous power

The lungs present nothing abnormal

The heart shows the apex in the fifth intercostal space, an inch outside the midclavicular line The borders are normal to the right and left The tones are regular and no murmurs are heard

The abdomen shows nothing of consequence and in particular no enlargement of the liver, and no tenderness over that organ

The extremities are negative

Reflexes—The patellar reflexes are doubtful It was noted in the examination of the eyes that the pupils reacted slowly to both light and accommodation

The interest in this case centers around the aneurysm, for it is hardly worth while to discuss the differential diagnosis, since there is scarcely the slightest doubt that we have a thoracic aneurysm before us With this in mind we took the patient to the fluoroscopic room and found as we usually do, that there was really more pathology in the chest than we had been led to expect The film (Fig 41) showed very beautifully a typical sacculated aneurysm either coming off the arch and involving the innominate or perhaps limited to this latter vessel only The fluoroscope, however, showed that there was a second mass

over to the left side posteriorly, and also showing a distinct pulsation expansile in character This latter could only be interpreted I think as a second aneurysm involving the descending part of the arch As I stated before, we nearly always find more pathology by the fluoroscope than our physical examination will lead us to suspect

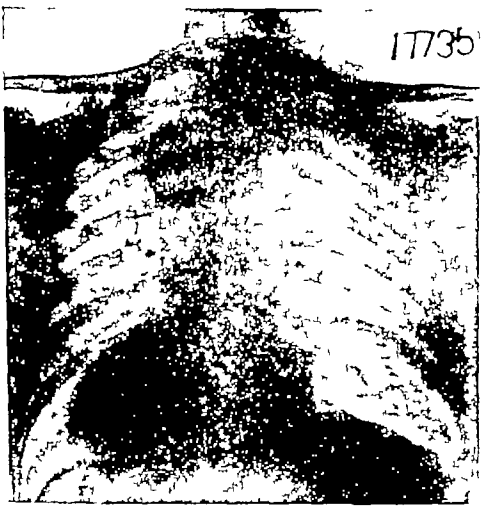

Fig 41 —Aneurysm probably involving the innominate as well as the arch Note the second mass on the left side (Postero-anterior view)

I think it may be useful in view of the 4 cases, to discuss some of the salient points of thoracic aneurysm in general in a rather didactic way

In regard to the classification of aneurysm we are not very much concerned so far as the clinical aspects of the thoracic aneurysms are concerned There are some points in the etiology which it might be well to stress All the studies of the last decade or two have served to emphasize the importance of syphilitic

aortitis as the leading cause of thoracic aneurysm I hardly think we are justified in saying that it is the only cause, because there are some other contributing factors These contributing factors are first certain infectious diseases, which may produce an arteritis and second, long continued strenuous work I have seen several aneurysms where no evidence of syphilis, either pathologically or clinically, could be discovered On the other hand, it is certainly true that the overwhelming majority are due to the combination of syphilis and excessively hard work This last operates undoubtedly through long continued raising of the blood-pressure It might be well to remind you that all physiologic experiments have shown that the aortic pressure may be raised to a point several times the normal height without producing perceptible stretching of the aorta With a diseased artery, where the resisting power of the blood-vessels to stretching is greatly impaired, this statement no longer holds true, and it is under these conditions that aneurysm develops, namely, considerably increased pressure with a greatly weakened arterial wall The statement, then, that syphilis is far and away the most common cause of aneurysm is merely another way of stating that syphilis is the most common cause of a type of arteritis of such a degree in the aorta as to make aneurysm possible

It might be noted that in the case of women in whom the factor of excessively hard work does not play so much of a rôle, a non-syphilitic aneurysm of the aorta is an exceedingly great rarity Indeed, thoracic aneurysm in women is so much less common than in men as to always attract attention In spite of this, we had a case recently of a thoracic aneurysm of the descending aorta in a colored woman Whether she had worked excessively hard or not I was unable to determine

With reference to the particular point of localization in the aorta, we may say, without going into the finer pathologic points, that the arch bears the brunt of all aneurysms—about 50 per cent of them being located there The common explanation for this is that the blood-current impinges most strongly on the aortic wall at the proximal end of the arch Next to the arch, the ascending aorta shows the largest number of cases,

approximately 30 per cent, and next to this the descending aorta, with the remaining 20 per cent A very interesting location, and one which is especially likely to remain entirely latent, is in the neighborhood of the sinuses of Valsalva Taken as a whole, thoracic aneurysm may be said to constitute approximately a half of all aneurysms We exclude from this account the small miliary aneurysms found in the lung in case of tuberculosis and similar conditions since these are not, properly speaking, objects of clinical investigation as such The same is true of the miliary aneurysms of the brain

It will be noted that the age incidence of aneurysm runs, broadly speaking, from perhaps thirty five to fifty five and the reason for this seems to me quite apparent A man is more apt to acquire syphilis early in life if he is going to acquire it and by this age it has had sufficient time to operate so that the aortic wall is thoroughly diseased, and in addition to this, he may have been working at strenuous work for enough years to produce the dilatation There are, however, conspicuous exceptions to this I have under my observation a young man in the thirties, who came into the hospital in a typical attack of angina pectoris and who gave a history of having acquired a lues probably at eighteen or nineteen, and of having done the most laborious work while serving with the Engineers during the World War To our surprise he presents a well-marked pulsation and unquestionable signs of an aneurysm of the descending aorta I mention this case as an exception and not a rule. I think there is another factor which should be emphasized and that is the question of adequate treatment of the syphilis The average individual who has acquired syphilis in the last ten years has generally had some treatment, albeit this may have been entirely inadequate The recent great influx of the negroes into our northern cities and especially a very large number of them who have worked on farms, has resulted in a very distinct increase in the frequency of aneurysm in our clinics in the last four or five years There is a very high incidence of syphilis among these colored workers, and it is rather exceptional to find one of them who has had anything which might be called adequate

treatment This great increase in a population showing a high incidence of untreated syphilis is, in my judgment, the reason for our marked increase of aneurysm incidence

Symptomatology—From a practical standpoint if anything is ever to be accomplished in the way of treatment the diagnosis must be made early, otherwise it is a perfectly hopeless outlook Our first concern, then, is to ask what are the early symptoms which might lead us reasonably to expect an aneurysm *I should like to emphasize the statement that there is no one early symptom* On the other hand, certain symptoms develop in a rather innocent way and do not of themselves suggest aneurysm at all One of these has become profoundly impressed on my mind because I have seen it a great many times, and some authors have gone so far as to say that it is the commonest early symptom I refer here to dyspnea of a paroxysmal type, sometimes simulating asthmatic attacks I have repeatedly seen a patient treated for asthma for months and months and in 1 case for a couple of years, when a good-sized aneurysm of the transverse arch was producing the whole trouble Even when the dyspnea is not paroxysmal in type and especially if it develops at night, one should think of aneurysm In my own experience I should say that pain is the most common initial symptom Anybody who comes to me with pain, with no obvious cause, if this pain be located in the chest and if the patient be anywhere around the aneurysm age, is subjected to an *x*-ray examination as well as a careful physical examination at once It would hardly seem necessary to remind you that the same process which produces aneurysm, produces also aortic insufficiency, so that any patient who comes to you with an aortic insufficiency of luetic origin should be subjected to a careful looking over to see if he has not an aneurysm in addition It would take us outside of the limitations of this clinic to refer to the old triad of aneurysm, aortic insufficiency, and cerebral spinal lues or tabes

I have already referred to the designation of the aneurysms of the ascending aorta and first part of the arch as aneurysms of physical signs These aneurysms produce well-marked physical

signs, such as dulness on percussion, heaving, expansile pulsation, and sometimes nothing else I well remember some years ago when one of my interns in a neighboring hospital asked me to step across to the other side of the ward to look at a case for an absent colleague This case presented a small, fluctuating tumor in the third right interspace It was about as big as a small mushroom and the pulsation in it was so slight that he had quite overlooked this and thought he had to do with an abscess of the chest wall He had a bistoury all ready to open it A careful physical examination, which was confirmed by the x ray, showed the patient to have an aneurysm the size of a small grapefruit, with a small prolongation which had gone right through the intercostal space, without producing much if any pain It was this little mushroom like projection which had brought him into the hospital, and the absence of symptoms had not caused the intern to suspect an aneurysm. This case was typical in that it shows how frequently all symptoms may be absent in cases where the aneurysm is located at this point Without going too much into the symptomatology we may say that the same thing applies to aneurysm of the descending aorta On the other hand a group of aneurysms referred to as aneurysm of symptoms are those which spring from the arch as it passes from front to back Aneurysms in this locality do not come in contact, at least in the early part of their growth, with the thoracic wall and hence physical signs are apt to be absent On the other hand, they come into relationship and press upon a series of structures, most of which have already been alluded to in the discussion of our cases They are especially the trachea, the primary bronchi, the recurrent laryngeal nerve, the esophagus, and the great vessels springing from the arch, or the great veins tributary to the superior cava If you take a good picture of the mediastinum or if you are sufficiently familiar with its anatomy to visualize it accurately, you will see that it is this group which can grow to a very considerable size without meeting any great resistance The pleura gives way rather readily to either side, and even the pericardium may be impinged upon to a considerable extent before the sac meets

with a serious resistance to its growth Furthermore, these tumors may grow upward toward the upper aperture of the thorax This explains why they may attain a very considerable size and produce a good many symptoms before they can be recognized by even a pretty careful physical examination It is in these cases that the x-ray proves invaluable There is another reason for keeping these cases in mind Postmortem statistics show that about 50 per cent of aneurysms die of an actual rupture of the sac, and may bleed so profusely as to die then and there I personally have seen on more than one occasion a rupture into the esophagus, into the bronchus, into the trachea, into the pleura, and the pericardium On two occasions at least I have seen aneurysms perforate the chest wall and bleed to death in that way A moment's reflection will show that the aneurysmal sac which is free to dilate without meeting serious resistance is the one most likely to rupture

One must be careful, however, not to attribute every expectoration of blood or every small hematemesis to an actual rupture of the sac In the further case-notes on two of the patients whom I presented today you will find that this very thing has happened In one of them, the tall colored man, the nurse on coming in in the morning found 2 or 3 ounces of blood in the form of a large dark clot on the bed alongside of him He was uncertain whether he had vomited or coughed it up, but further investigation made it pretty sure that it was a hematemesis He had had some little difficulty in swallowing and the probabilities are very strong that he had some ectatic veins in the esophagus due to the pressure of the sac on that organ The rupture of one of these veins is the probable cause of the hemorrhage One of the other patients, you will remember, had as almost his first symptom a couple of years ago the coughing up of some bright red blood Inasmuch as he steadily complained of dyspnea as his leading symptom, it is strongly probable that his hemorrhage came from some overdistended veins in the tracheal mucosa, or perhaps even lower down in one of the primary bronchi These two instances will serve to emphasize that a very considerable number of small hemor-

rhages are brought about in this way and are not due to an actual perforation of the aneurysmal sac itself From the standpoint of immediate prognosis this is a matter of considerable consequence Just at this point it may be wise to remind you that it is a very unsafe procedure to introduce a stomach tube or bougie of any kind in a patient complaining of difficulty in swallowing until you have made quite sure not only by physical examination but by the x-ray as well that no aneurysm is present During my student days I saw one of my most brilliant teachers neglect this precaution and directing his intern to bougie an individual complaining of difficulty in swallowing, had the mortification of seeing a tremendous gush of blood and the patient expire then and there Within the last few years a similar case has come under my own personal observation, though, fortunately, I was not actually present at the time of the accident

Perhaps as a closing word I can do no better than to remind you again that while the diagnosis of some aneurysms is exceedingly simple, the diagnosis of others is correspondingly difficult In every middle aged individual with obscure chest symptoms one should make a deliberate and careful examination of the chest both front and back for pulsation, a careful examination for the various physical signs I have enumerated, and a fluoroscopy should not be omitted It is only in this way that the cases, which are now distressingly frequent, of individuals with aneurysms who have gone for two and three years begging for a diagnosis will be eliminated

CLINIC OF DR. WALTER WILE HAMBURGER

MICHAEL REESE HOSPITAL

BUNDLE BRANCH BLOCK FOUR CASES OF INTRAVENTRICULAR BLOCK SHOWING SOME INTERESTING AND UNUSUAL CLINICAL FEATURES

THE following 4 cases of bundle branch block have occurred in a series of general cardiac patients covering a period of nine years During this period a considerably larger number of cases of intraventricular block have come under observation, but I have chosen to present these particular ones as each one presents a rather unusual clinical picture

The subject of bundle branch block is now about twenty years old, dating from the original experimental work of Eppinger and Rothberger in 1909 In this original work these authors "made a large number of experiments on dogs in which they injected solutions of silver nitrate into the ventricular muscle with the idea of determining the effects of destroying various portions of the muscle of these chambers on the electrocardiogram" A year later Eppinger and Stoerk were fortunate enough to observe two clinical cases the findings of which fitted in very well with the electrocardiographic findings in experimental animals Since this time a number of both experimental and clinical papers have appeared confirming and amplifying the original findings of Eppinger and Rothberger The current text books on cardiology are increasingly discussing this interesting pathologic entity

Just what do we mean by bundle branch block? When the muscle of the heart becomes affected by disease or other degenerative process, particularly if the disease process is chronic or associated with the production of fibrous tissue or other

organic changes, the integrity of the heart is thereby obviously impaired If this invasion of the heart muscle involves the conduction pathways of the ventricles, these changes may be observed in the electrocardiogram If these structural changes involve completely or incompletely one or the other or both of the branches of the bundle of His, they may be identified electrocardiographically The disease process may involve either branch completely or incompletely, interfering thus with the passage of impulses We speak of such interference as complete or incomplete bundle branch block If the pathologic changes occur in so-called "silent" areas of the heart, that is, away from the conduction pathways, the electrocardiogram may be normal or only slightly atypical Therefore the finding of bundle branch block should be interpreted as part of a more widespread process, that is to say, a process which involves not only the bundle branch, but likewise widespread areas throughout the heart muscle In other words, the finding of definite evidence of bundle branch block carries with it the implication that the heart is the seat of rather widespread degenerative changes

Disease of this type occurs usually in rather advanced years In 35 cases presented by Herrick and Smith, twenty of their patients were between twenty-five and sixty-five years, the youngest being twenty-three In Carter's series of 22 cases the majority of them were over fifty years of age, although he discussed a girl of sixteen, a woodcarver of twenty-eight, a gas-fitter of thirty-six, and so on

Etiologically, Herrick and Smith mention seventeen patients with an associated history of chronic nephritis, five with positive Wassermanns, ten with a history of acute rheumatic fever

From the standpoint of presenting symptoms, there is nothing particularly characteristic of patients suffering with bundle branch block Most of them have the usual complaints of advanced cardiac disease, namely, breathlessness on exertion, orthopnea, edema, often Cheyne-Stokes breathing, uremic manifestations, and the anginal type of heart-failure

From the standpoint of physical findings, up to recent times there have likewise been no characteristic data However,

King, in June, 1928, in a brilliant and illuminating paper on the "Clinical Recognition and Physical Signs of Bundle Branch Block," presents a series of 19 cases out of 100 which showed some very suggestive and striking findings King concluded that "the possibility of the clinical recognition of bundle branch block was suggested by the successful diagnosis in a patient with cardiac symptoms presenting (1) a visible bifid apex thrust, (2) feeble heart sounds with a sound and an asynchronous murmur accompanying the two elements of the systolic thrust." Herrick and Smith had previously commented on the finding of gallop rhythm and muffled first tones I feel the importance of King's findings are the recognition that as a result of a block of either the right or left branch, the normal synchrony of the two ventricles may be thereby disturbed, resulting in an asynchrony which may be great enough to enable one to demonstrate an asynchronous contraction of the two ventricles as evidenced by reduplication of the apex thrust by either inspection or palpation, by the presence of a gallop rhythm or by the finding of either a splitting or reduplication or muffling of the first tone at the apex with or without the accompaniment of a systolic murmur

With this brief introduction I should like to present the following cases

Case I. Advanced Permanent Right Bundle Branch Block with Delayed Auriculo-ventricular Conduction Time, Progressing to Complete Heart-block and Death.—This patient, M L fifty three years of age a clerk by occupation first entered the hospital October 9 1927 complaining of shortness of breath of two years duration easy fatigue for the past two years, and nocturia for five years He said that he had been in fairly good health until about two years ago at which time he developed shortness of breath During the past two years the dyspnea had become increasingly bothersome until at the present time he cannot continue his work, can practically climb no stairs, and feels uncomfortable on even the slightest exertion In addition to the dyspnea he complained of pain in the pit of the stomach which had been present for about one week, was of a depressing character and particularly noticeable upon coughing The pain had no relation to meals and did not radiate Cough dry and suffocating in character had been present intermittently for many years. His cough was occasionally associated with expectoration particularly during the course of an acute cold He had never coughed up blood The nocturia has been present two or three times a night for the

past five years As a child he suffered with measles, scarlet fever at fifteen years, pleurisy at twenty-two years of age He had no definite history of acute rheumatic fever, chorea, or tonsillitis

Physical examination revealed a slightly undernourished white male about fifty-five years of age, who did not appear acutely ill He was very slightly dyspneic and had a dry, suffocating cough with wheezing respirations There was some clubbing of the terminal phalanges There was slight prominence of the eyes, the pupils reacting promptly to light and accommodation Fine crackling râles were present at both bases toward the end of inspiration with musical, expiratory wheezing râles throughout the chest The apex beat of the heart was somewhat heaving in the fifth and sixth interspaces in the

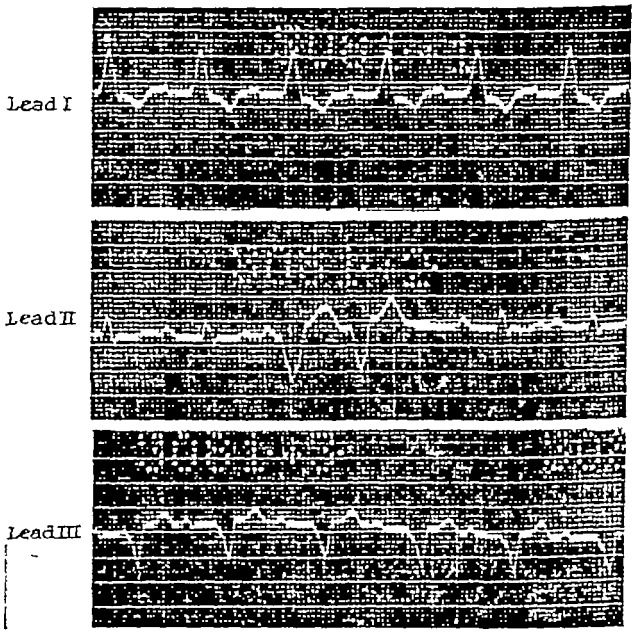

Fig 42 —Right bundle branch block. Frequent extrasystoles Depressed A-V conduction time, 0 28 sec , rate 83

anterior axillary line An arhythmia was present, apparently due to multiple extrasystoles The heart tones were soft and distant, and a presystolic murmur and thrill at the apex could be demonstrated The heart was enlarged both to the right and left with a total diameter of 19 cm The liver was one to two fingers below the right costal margin, slightly tender on pressure There was no demonstrable edema The impression of the senior intern was, "chronic myocarditis with beginning mitral failure, decompensation, mitral stenosis and insufficiency, cardiac asthma with chronic asthmatic bronchitis, mild emphysema, and prostatic enlargement "

An electrocardiogram (Fig 42) taken October 10, 1927 showed "marked myocardial involvement, right bundle branch block, frequent extrasystoles

arising from numerous foci at least two in the left ventricle one in the right ventricle and one in the auricle depressed auriculoventricular conduction time to 0 28 of a second sinus arhythmia rate 83 A two meter *x* ray heart plate showed the transverse heart diameter to be 17 6 cm with a transverse chest diameter of 24.3 cm The heart contour was laterally and anteriorly relatively normal

The laboratory findings showed hemoglobin 85 red count 4 660 000 white count 8000 The differential count showed 78 polymorphonuclears 22 small mononuclears The Wassermann and Kahn tests were negative The blood pressure systolic varied from 114 to 122 and diastolic from 58 to 78 The blood-chemistry was normal The basal metabolic rate on October 12th was 35 per cent. + On October 25th following bed rest and digitalis there was improvement in compensation and the basal metabolic rate was 23

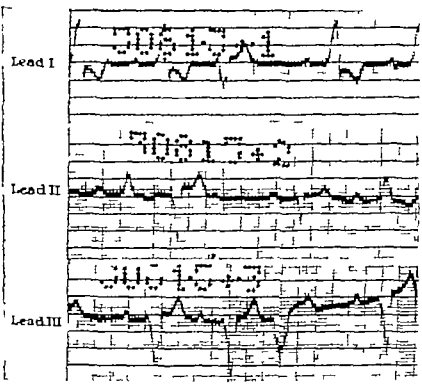

Fig 43 —This series essentially same as the first (Fig 42) Rate is slower (58) and there is an additional focus of extrasystole in the right ventricle P R 0.28 sec.

per cent. + Examination of the urine showed with the exception of a few hyaline casts and a faint trace of albumin nothing unusual

The electrocardiogram was repeated at various intervals during his hospital stay (Figs 43 44) and showed no striking change in the complexes a persistent right bundle branch block with delayed auriculoventricular conduction time up to 0 32 He left the hospital October 28th improved only to re-enter a year later in August 1928 with a recurrence of his previous symptoms with increasing dyspnea orthopnea edema of the legs and ankles with increasing enlargement and tenderness of the liver etc.

Examination on this second entrance showed substantially the same

findings as before with the exception of a general progression downward of all findings The electrocardiogram (Fig 45) on August 25, 1928 showed a

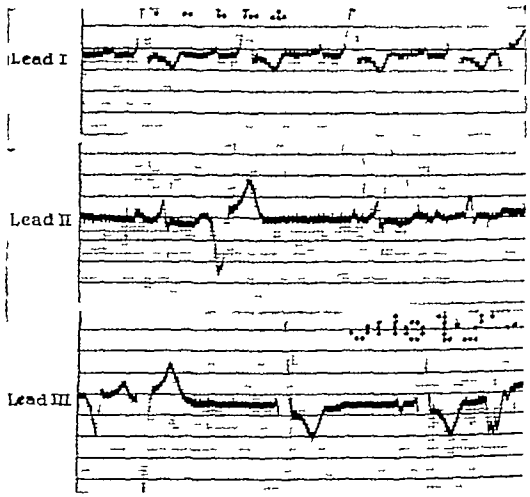

Fig 44 —A-V conduction still further depressed, 0 32 sec , rate 55 Most of the extrasystoles are left ventricular in origin arising from at least two foci

complete auriculoventricular heart-block with advanced block of the right branch as before In spite of various measures the patient became pro-

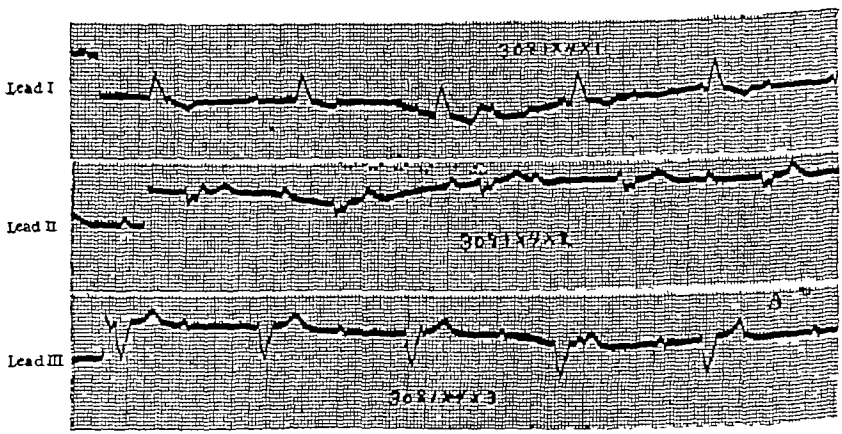

Fig 45 —Complete heart-block Advanced right bundle branch block The ventricular focus is in the right ventricle Auricular rate 62, ventricular rate 35

gressively worse and died August 31, 1928 Autopsy revealed a chronic mitral endocarditis with localized calcification and ulceration, mitral stenosis and

insufficiency chronic aortic endocarditis with calcification aortic stenosis and insufficiency hypertrophy and dilatation of the heart bilateral pulmonary congestion and edema chronic interstitial myocarditis

In review, one of the interesting features of this case is that for a period of ten months a progressive deterioration and degeneration of the conduction pathways developed as observed from time to time in the progressive alterations of the electrocardiogram. Starting with a right bundle branch block and a delay in the auriculoventricular conduction time, the degenerative process progressed until ten months later, when complete dissociation of the auricles and ventricles occurred, with continuation and progression of the blocking of the right bundle branch Likewise, in spite of any definite history of rheumatic fever, the findings at autopsy suggested the belief that the cause of this patient's cardiac disability was rheumatic This case is likewise of interest in showing that in the presence of pathology such as this, in spite of practically ideal care—the patient was extremely co-operative, was seen at frequent intervals, and had abundant rest and medication, that the progress of the cardiac degeneration could not be controlled or modified

Case II. Left Bundle Branch Block Following an Acute Coronary Occlusion of a Small Vessel Progressing to Auricular Fibrillation.—The second patient in this series concerns a physician fifty two years of age whom I have been seeing at intervals for nine years and whose findings I have presented on one or two other occasions for other reasons

On October 18 1920 at 11.30 o clock in the evening on a sleeper coming to Chicago he was suddenly seized with severe abdominal pain vomiting and diarrhea and believed he was the victim of so-called ptomain poisoning However he was somewhat concerned about himself and consulted me the following day upon his arrival in the city There were no striking physical findings which I could make out but upon having an electrocardiogram made (as I was somewhat dubious of the diagnosis of ptomain) his curve showed an unmistakable lesion of the left branch of the bundle of His giving a rather typical picture of left bundle branch block (Fig 46) My conclusion was that the so-called ptomain poisoning was actually the result of a small coronary occlusion involving the left branch of the bundle and resulting in this bizarre curve He made an uneventful recovery from this accident and disappeared from view for three or four years He was not seen again until 1926 when he came in because of slight shortness of breath irregularity of the pulse slight edema of the ankles. Examination revealed an unmistakable auricular fibrillation with early heart muscle failure and the electro-

cardiographic examination a few days later confirmed this opinion (Fig 47) The evidence of left bundle branch block still persisted, and the patient was cautioned to cut down materially on his professional activities and to lead

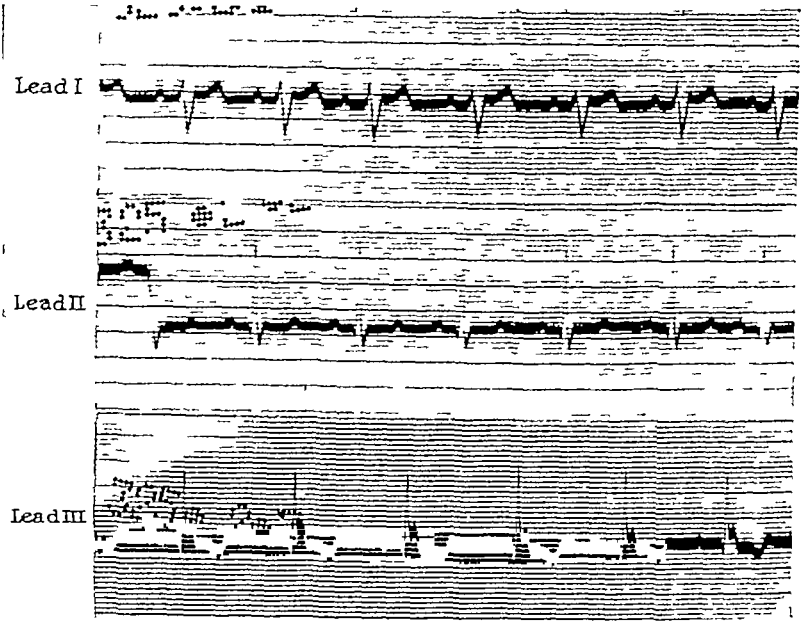

Fig 46 —Left bundle branch block following occlusion of a small coronary vessel, giving the clinical picture of "ptomain poisoning"

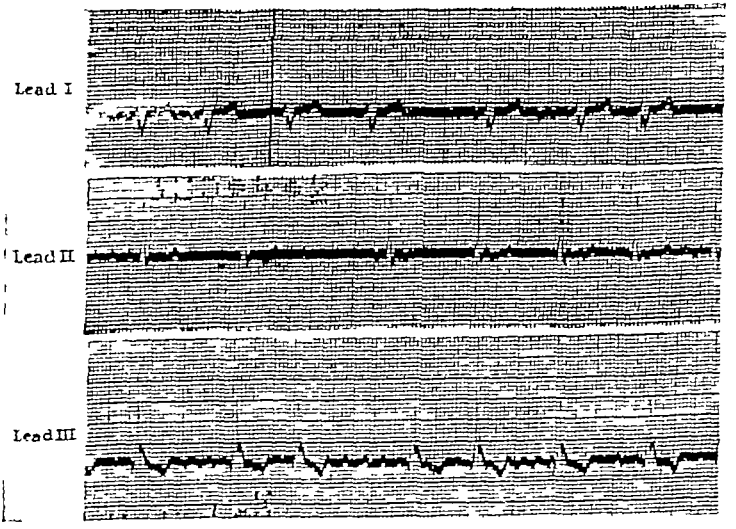

Fig 47 —Left bundle branch block and auricular fibrillation

so far as possible a quiet simple existence with moderation in diet etc. He again disappeared from observation until December 1927 at which time I was called in an emergency because of a sudden syncope with complete left sided hemiplegia from which he is now suffering During this past year he has been almost completely bedridden as the hemiplegia has made only very slight improvement and within recent months because of an increase in the signs of congestive heart failure he entered the hospital for more accurate study and observation

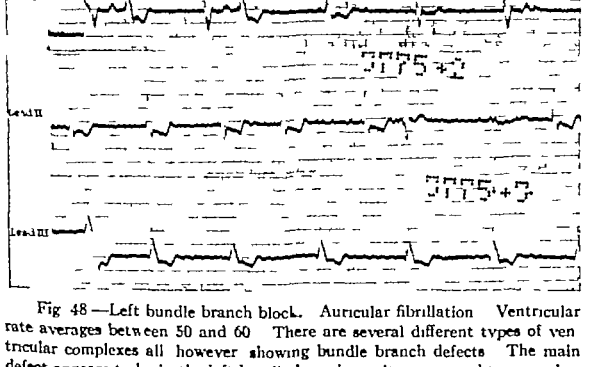

Fig 48—Left bundle branch block. Auricular fibrillation Ventricular rate averages between 50 and 60 There are several different types of ventricular complexes all however showing bundle branch defects The main defect appears to be in the left bundle branch resulting in a right preponderance. There are ectopic ventricular beats arising from two foci one in the right and one in the left ventricle

His heart now is enlarged irregular slow and still shows the findings of fibrillation and left bundle branch block as may be observed in his curve (Fig 48) In passing I may say that this patient is a member of a family with apparently poor cardiovascular tissue as several of his brothers have somewhat similar pathology an older brother having died within the past year of acute coronary thrombosis with angina.

In retrospect, this patient may be considered as one suffering with widespread cardiac pathology, initiated in a comparatively young individual by an attack of sudden coronary occlusion, occurring in a small coronary vessel resulting in left bundle branch block, progressing within six years to auricular fibrilla

tion, and finally resulting in cerebral embolism, probably from a mural thrombus in a fibrillating auricle, with left hemiplegia

Case III. Transient Right Bundle Branch Block from Digitalis, in a Case of Advanced Congestive Heart Failure.—The third patient I am presenting to show one of the effects of digitalis therapy, namely, the production of transient bundle branch block due to digitalis

This patient is a man of sixty-four, a merchant, who was brought to the hospital in an emergency with marked decompensation, cyanosis, orthopnea, distended and pulsating jugular veins, anasarca, Cheyne-Stokes breathing He gave a previous history of heart disease of several years' duration, and a past history of repeated attacks of acute rheumatic fever in his early years He entered the hospital in September, 1928 having been bedridden since

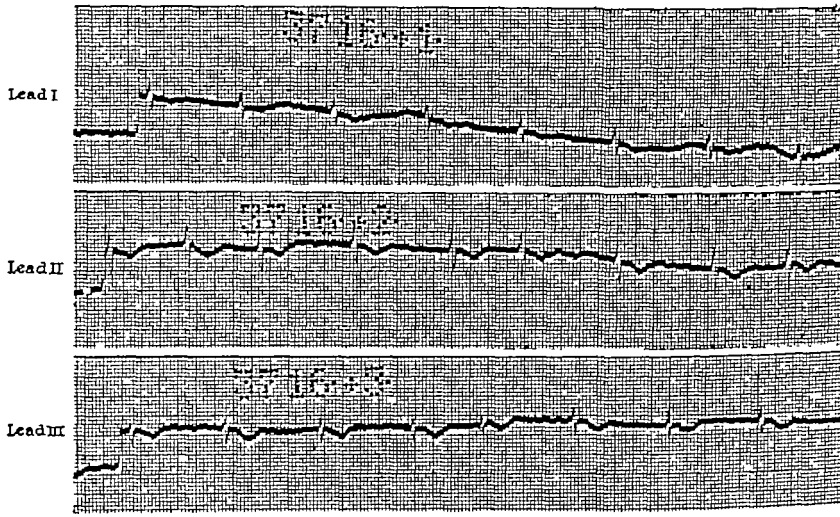

Fig 49.—Auricular fibrillation Rate 60 Inversion of T in all leads—probably a combined coronary and digitalis inversion

January of last year Examination revealed a rather obese, middle aged man with a heart enlarged to the right and left, the left border to the anterior axillary line The pulse was irregular, the apex was diffuse and only partially visible There were moist râles at both bases A harsh systolic murmur could be made out at both the mitral and aortic areas The abdomen showed marked ascites, a large tender liver, and in general the patient could be said to be critically ill I saw him a short time after entrance to the hospital and felt digitalis was urgently needed I ordered two ampules of digifolin intravenously every eight hours, with tincture of digitalis 2 drams by mouth every twenty-four hours

His first electrocardiogram (Fig 49) was made three days after digitalis had been given and showed auricular fibrillation with a rate of 60, inversion of T-wave in all leads Treatment was continued for another week with,

however diminishing amounts of digitalis because of some slight evidence of nausea

On September 14th his second electrocardiogram (Fig 50) showed an occasional atypical beat of the general configuration of right bundle branch

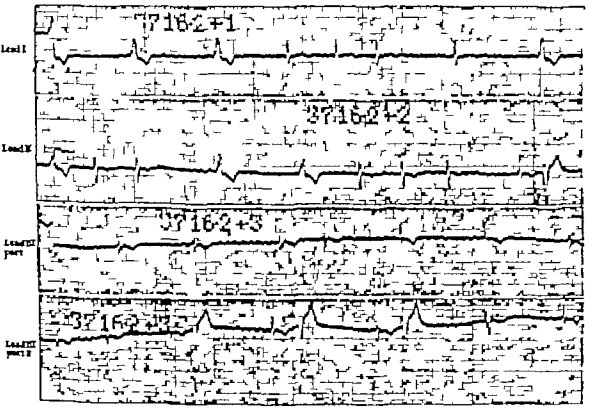

Fig 50 —Auricular fibrillation Recurrent extrasystoles from multiple foci from both right and left ventricles Alternating right bundle branch block (?)

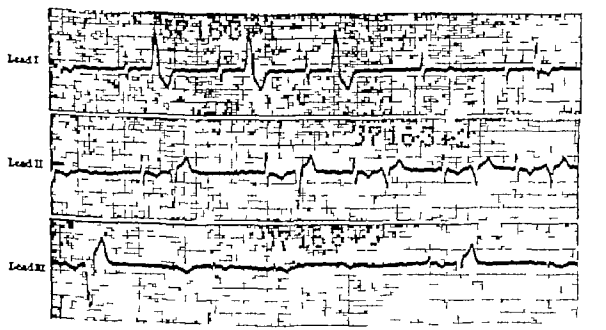

Fig 51 —Same as Fig 50

block On September 18th, with the discontinuance of digitalis, his third electrocardiogram showed the continued presence of these beats in many leads, alternating with normal ventricular complexes (Fig 51) The finding of alternating right bundle branch block complexes has been observed before and described recently by Leinbach and White as "two to one right bundle branch block," that is to say, each alternate conduction in the right branch of the bundle is interfered with, giving so called two to one right bundle branch block During subsequent days, with complete stoppage of digitalis, the heart gradually returned to its original state, although compensation was not completely restored On September 25th (Fig 52) one sees complete disappearance of the evidence of bundle branch block and only the finding of fibrillation persists In other words, I feel that this case should be considered a case of advanced, senile, degenerative, cardiopathy with congestive

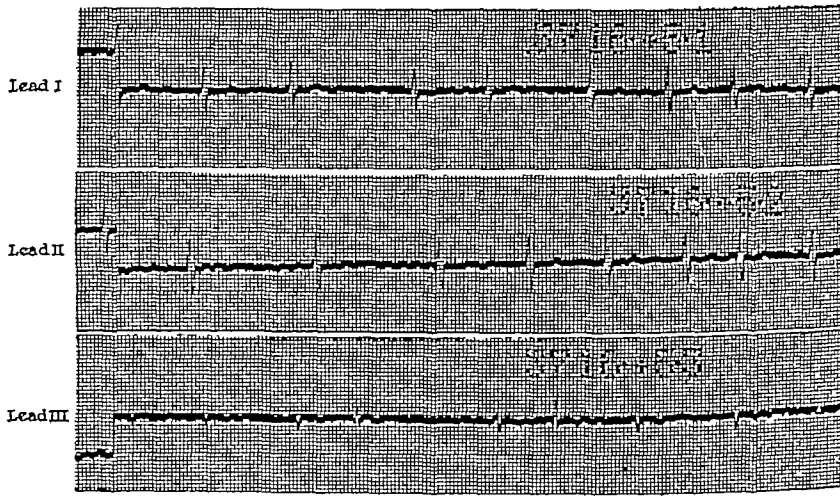

Fig 52 —Same as Fig 49, except for upright T waves

heart-failure, generalized anasarca, cyanosis, orthopnea, rapid fibrillation, with prompt response to massive digitalis therapy, with the development of right bundle branch block and at times two to one block, later with complete disappearance of the interference with the conduction pathways, with the omission of digitalis

While it is generally known that digitalis is able to produce delayed auriculoventricular conduction time, and even complete auriculoventricular dissociation, and at times incomplete bundle branch block, this is the first case, so far as I am aware, where the definite finding of transient bundle branch block due to digitalis has been described

Case IV Acute Severe Right Bundle Branch Block with Auricular Tachycardia in a Young Boy, the Result of an Acute Upper Respiratory Infection.—My fourth case is perhaps the most interesting of all It concerns a young boy of four and one half years incidentally the youngest case of bundle branch block that I have been able to find This child was seen in consultation with Dr Mark Jampolis and entered the hospital December 3 1928 with the history that he was well until about five days before entrance, when he awoke with a stomachache The child remained in bed that day and part of the following day after which he felt better for a few days and then again complained of palpitation vague aches and pains in the extremities and back, and during the second attack he vomited During all of this time his temperature remained normal Two or three days following this he felt better and remained out of bed The morning of admission to the hospital he awoke again complaining of palpitation The mother states that during these attacks of palpitation he has difficulty in breathing He does not become cyanotic but does become pale The mother states that the child has always been a nervous and irritable youngster and that during the past year on several occasions he has complained of a similar rapid beating of his heart. There is no history of convulsions His appetite has been only fairly good and his bowels regular He was a full term normal child breast fed until six months walked and talked at fourteen months In the family history there is nothing significant At six months he had so-called bronchitis

I saw him with Dr Jampolis at 5 o clock in the afternoon of his admission to the hospital At the time of examination his heart was not enlarged the right border was 3.5 cm. and the left 6 5 cm. from the midsternal line and about ½ inch within the left nipple line The lungs were entirely clear When my stethoscope was first placed over his heart the heart tones were normal the rate was regular and slow about 80 per minute, but while listening the heart suddenly jumped to a rate as nearly as I could count of 240 per minute After two or three minutes at this rate it again became slow only to return to its tachycardia My note stated as follows The rate now varies from 240 to 120 There are occasional groups of three or four beats with a pause and then four or five beats with a pause There is some evidence from the grouping of beats and pauses of some relationship to the respiratory cycle. I put down as my impression after examination that the sudden onset and stopping of the tachycardia suggests true paroxysmal tachycardia. The sudden change to slow irregular rhythm with dropped beats suggests partial block. I however cannot absolutely rule out impure flutter Must await electrocardiographic evidence Believe the arhythmia is not serious but is toxic in origin Advise quinidin ice bag to precordia and so on

I should have stated that on the afternoon of admission at 3 o clock his rectal temperature was 100 6 F at 8 101.2° F and at 9 o clock 103 F So far as his mother knew this was the first temperature that the child had had His white count was 13 700

The electrocardiogram taken about 5 P M on the day of admission (Figs 53–55) showed a large number of unusual and bizarre findings exceedingly difficult to interpret but which might be discussed as follows

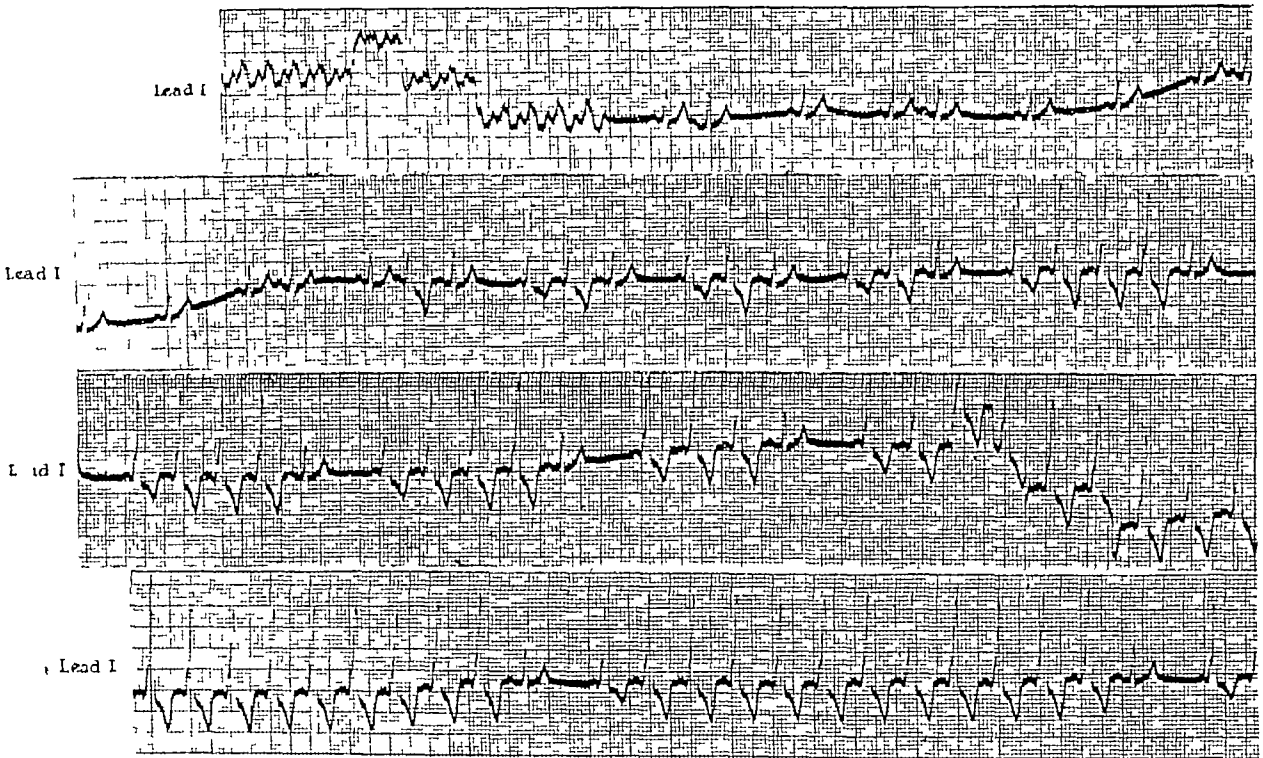

Fig 53 —At the beginning of lead I there is a ventricular tachycardia with a rate of 214 This ceases abruptly and gives place to a sinus rhythm with a rate of 68 This rhythm, however, is interrupted by frequent auricular ectopic beats This ectopic focus finally takes over the function of pacemaker and at the same time a right bundle branch block develops At first the ventricular conduction follows the normal pathway at every third cycle, then at every fourth or fifth cycle, then only occasionally

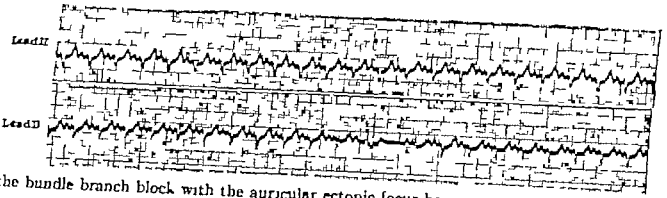

Fig 54.—Finally the bundle branch block with the auricular ectopic focus becomes completely established at a rate of 136

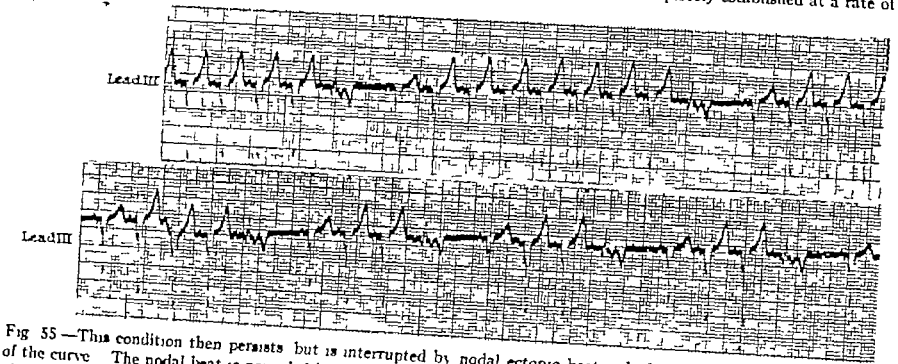

Fig 55.—This condition then persists but is interrupted by nodal ectopic beats which become more frequent toward the end of the curve. The nodal beat is preceded by a P wave of the same character associated with the cycles showing bundle branch block. This auricular impulse however is blocked at the node and the nodal ectopic beat results. The impulse from the nodal focus spreads through the ventricles in a more nearly normal

At the beginning of lead I there is a ventricular tachycardia with a rate of 214 This ceases abruptly and gives place to a sinus rhythm with a rate of 68 This rhythm, however, is interrupted by frequent auricular ectopic beats This ectopic focus finally takes over the function of pacemaker and at the same time a right bundle branch block develops At first the ventricular conduction follows the normal pathway at every third cycle, then at every fourth or fifth cycle, then only occasionally Finally the bundle branch block with the auricular ectopic focus becomes completely established at a rate of 136 This condition then persists, but is interrupted by nodal ectopic beats which become more frequent toward the end of the curve The nodal beat is preceded by a P wave of the same character associated with the cycles showing bundle branch block This auricular impulse, however, is blocked at the node and the nodal ectopic beat results The impulse from the nodal focus spreads through the ventricles in a more nearly normal fashion than that from the ectopic auricular focus

Summing up there is present ventricular tachycardia, sinus rhythm, auricular ectopic beats, intermittent bundle branch block, nodal ectopic beats

The evening of admission at 7 15 o'clock, 1/5 grain of quinidin having been given by mouth, the heart rate was recorded as 240, then dropped to 120, and thirty seconds later again went back to 240, with apparently regular rhythm At 8 45 the same night after nearly a grain of quinidin had been taken the heart rate remained at 220 for two minutes, slowed suddenly for five or six beats and then returned to its rapid rate

The early morning notes on the fourth record "a temperature of 103° F during the night, in the morning 101° F The patient spent a restless night, but is more comfortable now, heart rate is 120 and regular" My note at 11 o'clock in the morning of the fourth states "The patient seems much improved Heart regular, rate 120 Right border not as far out as before, only 2 cm from midsternal line Lungs contain roughened breath sounds with coarse crepitant rales I suggest ½ grain of quinidin every three hours"

On the night of the 4th at 6 o'clock the note was made "The patient more comfortable, heart rate 110 No suggestive symptoms of rapid heart throughout day" On the morning of the 5th, "patient more comfortable, heart rapid, regular, 110 Temperature range lower, 99 8° to 100 2° F Throat slightly reddened, coughs infrequently, heart borders within normal limits" The following day, "pulse 96 with respiratory arhythmia, slightly accentuated second aortic tone"

From this time on, patient made an uneventful recovery and left the hospital a few days later with instructions to remain at complete rest in bed A second electrocardiogram taken the morning of the 4th (Fig 56) shows a perfectly regular rhythm, rate 115, with typical right bundle branch block with ectopic auricular tachycardia

Another electrocardiogram was taken on the 5th (Fig 57) showing right bundle branch block with ectopic auricular tachycardia, rate 107, P-R interval 0 08 second The amplitude of all complexes is increasing as compared with the immediately preceding curve On the following day, the 6th, a fourth curve (Fig 58) showed the same as the one immediately preceding except for irregularity in rhythm, probably vagal in origin

Since leaving the hospital the patient has remained for the most part in bed but has been gradually allowed to get up He is entirely free from symp-

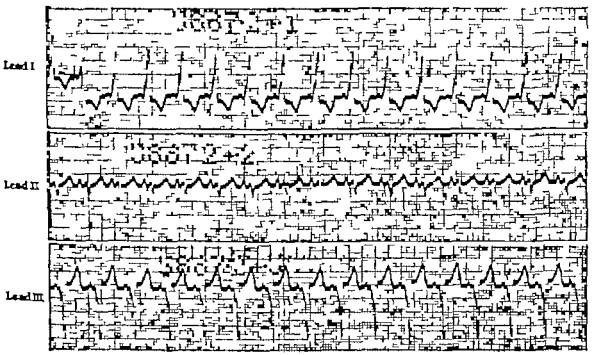

Fig 56—Right bundle branch block with ectopic auricular tachycardia. Rate 115 P R interval 0 08 sec.

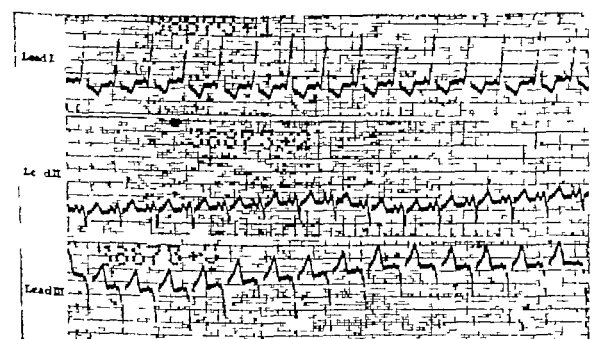

Fig 57—Right bundle branch block with ectopic auricular tachycardia. Rate 107 P R interval 0 08 sec. The amplitude of all deflections is increased as compared with the immediately preceding curve

toms eating sleeping and looking well and so far as his mother can determine, quite himself Upon request he returned to the hospital a week ago for

a repeat electrocardiogram (Fig 59) which shows substantially the same as the preceding curve, with a continuation and persistence of the right bundle branch block A teleoroentgenogram (Fig 60) taken at this time shows the heart contour to be somewhat globular suggesting a left auricular and ventricular increase

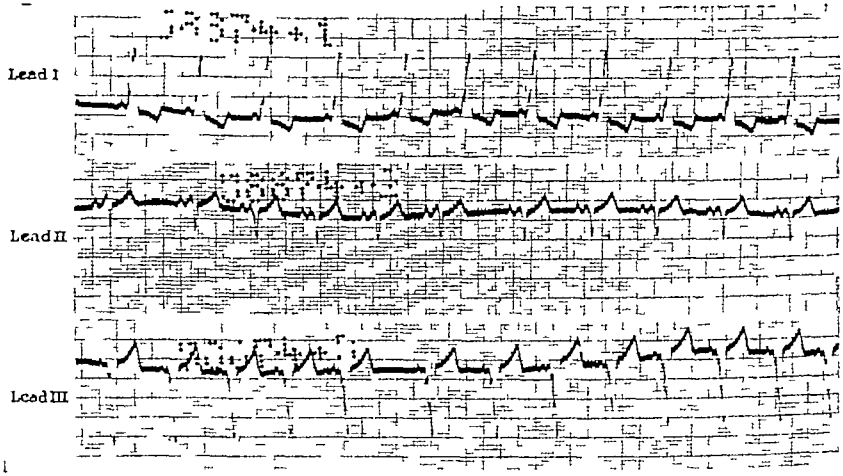

Fig 58 —This curve is the same as the one immediately preceding except for an irregularity in rhythm probably vagal in origin Rate 100

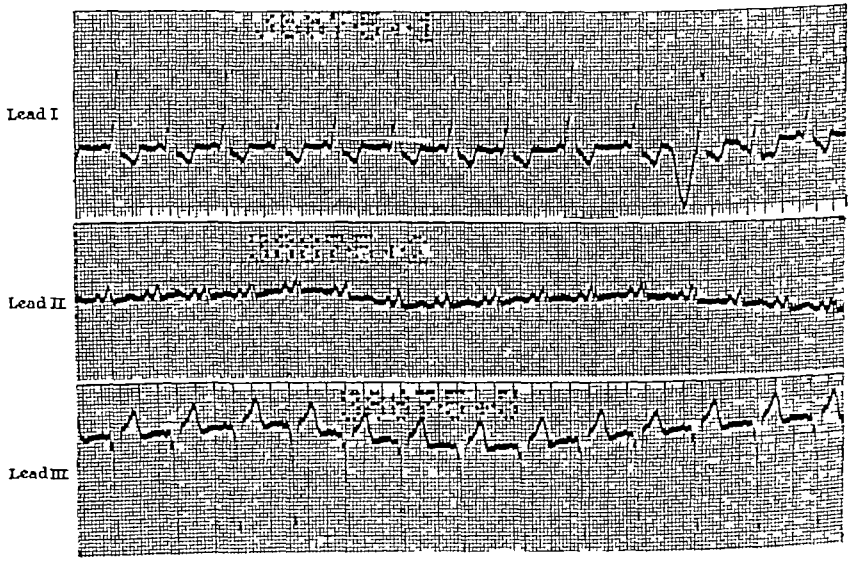

Fig 59 —Sinus arhythmia with right bundle branch block The rate varies from 100 to 120 Marked left ventricular preponderance (Judging from previous curves it is possible that auricular focus is outside of the sinus)

Fig 60 —Teleoroentgenogram of heart of four and a half year old boy with right bundle branch block the result of an acute upper respiratory infection (flu) Heart contour appears somewhat globular suggests a left auricular and ventricular increase.

Summing up this case, it appears to me to be an example of an acute severe right bundle branch block with an ectopic auricular tachycardia occurring in a young boy, four and a half years old, the result of an acute respiratory infection (grip), although at the onset he was free from fever His course in the hospital with reddened throat, cough, crepitant râles, temperature to 103° F, occurring during our present "influenza" epidemic, and in view of somewhat similar examples of arhythmia, tachycardia, and partial block which I described a number of years ago in connection with other respiratory infections, makes me conclude that this unusual involvement of his heart was part and parcel of his grip infection His last curve taken approximately six weeks after the onset, showing a persistence of the right bundle branch involvement, makes me

believe that at least for the present this damage to the heart will be more or less permanent, although for a period of three to six or nine months one cannot positively determine this It is interesting to consider that occasionally one of the points of attack of the influenza toxin is the conduction pathways of the heart, as evidenced by this case, but just how seriously and permanently this particular heart is damaged only the future can decide

In conclusion, these 4 cases may be briefly said to be

1 Advanced permanent right bundle branch block with delayed auriculoventricular conduction time, progressing to complete heart block and death

2 Left bundle branch block following an acute coronary occlusion of a small vessel progressing to auricular fibrillation

3 Transient right bundle branch block from digitalis in a case of advanced congestive heart failure

4 Acute severe right bundle branch block with auricular tachycardia in a young boy, the result of an acute upper respiratory infection

CLINIC OF DR ROBERT W KEETON

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OBJECTS AND METHODS OF TREATING PNEUMONIA

PNEUMONIA is a very discouraging disease to treat In a large general hospital, where there are many medical services, many variations in the orders outlined for the treatment of these cases, and a wide variety of interns in charge, the mortality is often surprisingly constant In fact this mortality seems to depend largely on the nature and virulence of the etiologic agent rather than on the methods used to combat its progress One can understand, therefore, why many physicians have adopted a fatalistic philosophic attitude toward a case of pneumonia The physician is going to do everything that he can for the patient, and he is perfectly willing to have the family suggest consultation, but he is convinced that the recovery of the patient depends on factors over which neither he nor the consultant have the slightest control

It is true that there exists a specific treatment for one group of cases However, this group is relatively small as compared with the sum total of all pneumonia cases, and the conditions requisite for the use of this specific treatment are such that it has not become available for the unhospitalized patient

A very large amount of energy has been consumed in the unsuccessful search for specific therapy in other types of the disease Indeed, as we are coming more and more to appreciate the mutations of the causative agents and their ability to adopt measures against the immune bodies of the patient, one grows rather pessimistic of the success of specific therapy

If one looks back a little into the history of the treatment of typhoid fever, he will find that many of the methods (protein reactions, vaccine injections, etc) now under experimental use in pneumonia, were applied to it and abandoned This disease was conquered by prevention and by a rational method, first clearly outlined by Coleman,[1] of protecting the patient against the invading typhoid organism until he had sufficient time to develop an immunity

It would seem therefore that more energy should be used in studying the morbid physiology of the pneumonia patient If this is understood, we might be able to protect the patient until his immunity is sufficient for recovery If we are successful in protecting a typhoid patient for three weeks, it would seem that with more intensive effort, a pneumonia patient could be protected over five to nine days

I wish to discuss the very meager information that we possess as to the physiologic perversions in pneumonia and to cite 3 cases successfully treated

Metabolic Requirements—DuBois[2] has shown that the metabolic requirements for patients are governed by certain general laws irrespective of the nature of the agent causing the temperature Thus for each degree rise in temperature (Fahrenheit), the metabolism is increased 7 2 per cent Restlessness increases it 10 to 30 per cent and toxemia (a term which is in good standing clinically but which has not been defined physiologically) 10 per cent The metabolism in pneumonia behaves much as it does in a severe case of typhoid So if one wishes to prevent significant loss in body nitrogen and body weight, the patient must be fed a diet containing approximately 100 per cent more calories than would be required for the same patient under basal conditions, in other words, basal + 100 per cent

In view of the short duration of the disease, most writers consider that it is neither necessary nor important to feed pneumonia patients a diet sufficient to protect their body weight and nitrogen As a matter of fact, the influence of this measure on the behavior of a group of pneumonia cases has not been rigidly tested out It is easily appreciated by all that the diffi-

culties of feeding a desperately sick patient such large quantities of food may be great or even prohibitive

On theoretic grounds alone and from the consideration of the beneficial effects of food on typhoid patients, one would conclude that the ideal pneumonia diet would be one approximating the metabolic requirements

The Local Pulmonary Condition—There appears to be two very different types of pulmonary reactions, one typified by the classical lobar pneumonia[3] and the other by the influenzal bronchopneumonia In the lobar type the pathologic process is a local one which may be more or less extensive The remainder of the lung remains normal and retains its normal capacity for exchange of oxygen and carbon dioxid In certain states of hepatization this affected portion of lung is both airless and bloodless and is excluded from the circulation Hence in many of these patients there is no interference with the oxygen saturation or load in the blood It is therefore obvious that the distant cells would not develop an oxygen want and would not then develop perversions of the metabolism due to this cause

In the case of the influenzal bronchopneumonia,[3, 4] the pathology resembles that found in gas poisoning and the pulmonary edema stage of the lobar pneumonia There is a diffuse bronchial reactions which may be of an anaphylactic nature, and here and there where the drainage is poorest and the reaction is greatest, a patch of consolidation appears As time goes on these patches may increase in size and meet each other Then a diagnosis of confluent bronchopneumonia is made It is quite obvious that from the very onset of this disease, the physiologic exchange of oxygen and carbon dioxid is upset The whole lung is involved, and shortly after the onset of the disease, cyanosis manifests itself The alveolar membrane has become relatively impervious to the oxygen The blood is snatched away before it has time to acquire its normal load of oxygen If these bloods are equilibrated with oxygen, they are found to have a normal oxygen capacity This means that all the outlying tissues of the body are subjected to oxygen want Oxygen want results in excess acid production and makes the

blood a poorer transport mechanism for acids, hence allowing their accumulation in the tissues When the extent of these changes on the various types of body cells have been analyzed, we will doubtless then understand what is going on in the patient when the clinician tells us that he has a high degree of toxemia

The Circulation—From the inspection of a pneumonia patient, one is apt to develop a wrong conception of the circulatory difficulties One sees a cyanotic patient, finds one or more lobes consolidated, hears a snappy second pulmonic tone, and he visualizes a heart which is working under great strain and having great difficulty in keeping the load of blood moving through the lungs Then one observes a capillary pulsation in the nails and a rosy cheek, and he makes the mental note that there is a paresis of the vasomotor nerves and center

As a matter of fact, the cyanosis does not indicate poor cardiac action,[8] and the cardiac failure arises from an inadequate blood volume rather than a pulmonary obstruction

The capillary disturbances in turn are due to a toxic effect on the capillaries themselves Perhaps it will be helpful to develop this picture with more detail If a man is suddenly transported into a high altitude or subjected to a decreased oxygen pressure, there is an immediate increase in the number of red blood-cells with a corresponding decrease in the fluid content of the blood If the individual corpuscles cannot obtain their maximum load of oxygen, then the total amount of oxygen transported can be increased only by increasing the individual number of carriers This then is a compensatory reaction of anoxemia, of oxygen want If now there should be liberated into the blood-stream capillary poisons similar to the well-known histamin hydrochlorid, then there would be a further tendency for the blood to lose its fluids into the tissues and into the lungs, in the latter case, producing a pulmonary edema This would result in a concentrated blood with a hemoglobin content 100, 110, 125 If now these capillary poisons should cause a larger number of extra capillaries to open up and increase the size of the vascular bed, some 20 to 50 per cent , it can be

seen that the blood volume absolutely reduced would now be still further relatively reduced and be rendered further in adequate

The author[5] in 1918 discussed the circulatory state of the influenzal pneumonia patients, and described it as due to toxemia shock. Underhill and Ringer[4] in 1920 reported an actual concentration of the blood in the more severe cases The heart therefore fails because it is attempting to keep a highly concentrated fluid with increased viscosity moving about efficiently in a bed 20 to 50 per cent too large for it

The Outlying Tissues—As previously stated one of the most obvious effects of an anoxemia is excess acid production and a tissue retention of these acids because the transport capacity of the blood is reduced Palmer[6] has shown that such an excess acid production does actually occur This fact is firmly established in the clinicians' mind

The Liver—The destructive effects of the pneumonic process on the liver are great

Postmortem, a wide variety of changes have been described (perihepatitis, cholangitis, parenchymatous hepatitis, fatty changes, nutmeg liver, and cirrhosis) The frequency of a frank jaundice in these cases is familiar to all, and there is a still larger group of patients with a subicteric tint to the scleræ, who doubtless have a jaundice which blood examination would reveal More recently Harris[7] has subjected a number of cases of lobar pneumonia, in which there was no increase of bilirubin in the blood, to liver function tests, and found that in all of these cases there was evidence of liver damage In a corresponding group of other infections (typhoid, scarlet fever, erysipelas) such damage did not occur

To the liver has been ascribed a detoxicating function It detoxicates bacterial poisons whether they be elaborated within the organism or in the gastro-intestional tract It also has a relatively important phagocytic function

More recently, Andrews[8] and associates have shown that urinary proteins may be of liver origin These proteins have been split off from the liver as the result of liver injury Since

this molecule is a foreign protein in the blood-stream, it is eliminated by the kidneys

Mason[9] has shown that if a few grams of liver tissue are dropped into the peritoneal cavity of the dog, a fatal toxemia (eighteen to twenty-four hours) results This same result has been obtained by Andrews when the liver is transplanted into the axilla The corrosion specimens of the liver in cirrhosis prepared by McIndoe,[10] render it probable that cholemia appears when the hepatic artery is no longer able to supply the liver parenchyma with blood By analogy one might consider that this severe intoxication described by Mason following the autolysis of liver tissue is similar to the intoxication of liver insufficiency seen in cholemia

If we now consider the pneumonia patient and follow the clinical course of the disease, when "the toxemia" becomes manifest, we find many points of similarity to the clinical picture produced in liver insufficiency The urine contains albumin, and the circulation rapidly develops the shock state discussed in a previous section It therefore seemed to me that if the therapy in pneumonia were directed toward the prevention of liver damage that the course of the disease might be influenced It is thoroughly established clinically and experimentally that glucose given in large quantities exerts a powerful protective influence in the presence of liver damage Hence it was decided to protect the patient with an adequate quantity of glucose and an abundance of fluids

In the tables, the glucose is figured as the total available glucose from all sources All foods fed the patients were weighed and the glucose equivalent estimated

Case I.—The patient was white, male, age sixty seven years, who entered the hospital within twelve hours of the onset of bronchopneumonia, with major findings in right lower lobe He was severely prostrated and cyanosis was established on admission

Past History—Duodenal ulcer, controlled by medical management Red blood-cells, 4,320,000, white blood-cells, 12,500, hemoglobin 70 Differential moderate, increase in polymorphonuclear leukocytes

Urine—Trace of albumin—sugar and acetone—negative Microscopically a few granular and hyaline casts

Sputum —Prune juice to bright frothy red numerous streptococci and pneumococci

Clinical Course —During the first three days every effort was made to feed the patient at two hour intervals because of his duodenal ulcer and to push fluids because of the febrile state However he rapidly developed difficulty in eating He was so weak, listless and dyspneic that it was impossible to force the food on him By the third day abdominal distention had become marked and it was impossible to get him to take water in appreciable quantities Consequently fluid intake by mouth was supplemented by the subcutaneous administration of 5 per cent glucose 1000 c.c at a time To this solution a small amount of novocain was added so that the patient experienced little actual pain during its administration Below is attached a summarized table showing the progress of the case the quantity of fluids taken and glucose injected It will be noted that the fluids given were usually 3000 c.c or better and the glucose amounted to 100 gm per day

TABLE 1

White—Male. *Age sixty seven years*

Date.	Temperature.	Pulse.	Respiration.	Fluids mouth	Fluids, skin.	Total.	Glucose.	Calories.	Urine.
November 20th—Onset of bronchopneumonia									
21	102 4	120	36						
	101 6	84	32	2970		2970	81	1500	
22	102 2	120	40						
	100 2	108	32	3390		3390	61	1130	790
23	101	134	28						
	100 2	112	22	1950		1950	51	550	1200
24	100 6	140	36						
	98 8	110	23	1500	1000	2500	86	570	815
25*	99	120	36						
	98 2	106	26	1530	2000	3530	100	400	680
26*	99 4	120	32						
	98 6	106	24	340	2000	2340	100	400	1050
27	99 6	112	28						
	98 2	100	24	1500	2000	3500	100	400	870
28	99 6	104	32						
	98 4	96	24	1190	2000	3190	100	400	1350
29	99 8	100	32						
	98 6	90	24	1140	1000	2140	186	850	1135
December 20th—Discharged in ambulance									

* Very weak oxygen inhalations at irregular intervals Patient uncooperative in its use.

As soon as this regime was well established the improvement of the patient was definite and marked After November 29th he was able to take

sufficient food and fluids by mouth If he had a poor day and did not receive his quota, an hypodermoclysis was given at bedtime

In addition to the above management morphin was used freely Caffein sodium benzoate and digifolin were used for stimulation

Case II —A white female aged fifty years developed a chill at 4 P M, and entered the hospital at 10 P M on November 27, 1928 A diagnosis of right lower lobar pneumonia was made Prostration was severe and cyanosis appeared early

Red blood-cells, 4,150,000, white blood-cells, 14,000, hemoglobin 65

Differential—preponderance of polymorphonuclear leukocytes

Urine —Trace of albumin, otherwise negative

Sputum —Absent

TABLE 2

White—Female *Age fifty years*

Date	Temperature	Pulse	Respiration	Fluids, mouth.	Fluids, skin	Total	Glucose.	Calories.	Urine.
November 27th—Onset with chill									
11/28	103 6	100	40						
	101 4	95	32	3530		3530	83	409	600
11/29	103 4	100	38						
	102	95	28	3315		3315	88	673	950
11/30	103 6	95	30						
	101 8	95	28	3515		3515	75	826	750
12/1*	102 8	110	30						
	101	100	30	3525		3525	111	826	1100
12/2*	102 4	100	30						
	101 4	98	28	1380	2000	3380	175	863	2125
12/3*	102 4	95	28						
	100 6	88	22	355	2000	2355	110	440	2700
12/4*	101	84	22						
	99 2	78	22	725	2000	2725	149	663	1175
12/5*	99 8	84	20						
	98	78	18	1120	3000	4120	200	908	560
12/6	99 4	80	24						
	99	70	20	1600	1000	2600	175	1035	2200
12/7	99 6	82	24						
	98 4	78	20	2700		2700	162	1039	2300
12/8	99 4	80	22						
	98 4	74	20	3775		3775	206	1283	1515

* Period of complete disorientation Marked toxemia

Clinical Course —During the first four days in the hospital an attempt was made to give the patient 3000 c c of fluids and 150 gm of glucose by mouth It will be noted that the glucose taken was approximately half of

the amount ordered The fluid intake was well maintained The cyanosis became more pronounced and the state of the circulation was more unsatisfactory On the night of December 1st delirium began and continued until December 5th During this time she was entirely disoriented and almost unmanageable Almost everything given by mouth was refused Beginning December 2d 1000 c.c. 5 per cent. glucose was given subcutaneously every eight hours until the patient cleared on December 5th and again began to co-operate

The improvement in the toxemia and delirium was again marked after administration of the glucose at these regular intervals On the morning of the 5th the temperature reached a level of 99 4 F and after this showed no significant rise

Her further recovery was uneventful Medication in this case consisted of bromids per rectum for restlessness Morphin for rest Caffein sodium benzoate and digifolin as needed for stimulation

Case III.—This is a white female age thirty two years who two and a half years ago developed an eclampsia and was delivered of a child by cesarean section Two months prior to her pneumonia she was delivered of a second child by cesarean section She had a double mitral lesion and following the second cesarean section she developed a definite decompensation with moderate edema of ankles liver below the umbilicus and moderate ascites She was recovering from the decompensation when she suddenly developed a pain in right chest associated with a friction rub and blood tinged sputum. When first seen I was unable to determine whether she had thrown off an auricular blood-clot or had developed a true pneumonia However she has run the clinical course of a severe pneumonia Two days prior to the onset of the pneumonia, the urine contained large quantities of albumin and many renal cells but no casts. The blood pressure was 156/90

On admission Red blood-cells 4 100 000 white blood-cells 11 500 hemoglobin 65

Differential slight preponderance of polymorphonuclear leukocytes

It seemed to me that in this case where there already was presumably liver damage due to the old eclampsia and the recent cardiac decompensation that there was every reason to crowd the quantity of glucose to the limit

The pneumonia began December 30th and patient entered hospital on December 31st so that intensive treatment was begun within twenty four hours of the onset of the disease During the first three days we were handicapped by vomiting but this stopped during the day of January 2d and has not returned However we have had considerable difficulty with abdominal distention which is presumably aggravated by the large quantities of carbohydrates in the diet At the onset the patient was given fluids subcutaneously four times On the first two occasions, she absorbed these fairly well On the last two occasions quite poorly In fact we apparently overstepped the traffic limit of her tissues for water as the face became somewhat edematous. At this stage euphyllin was started and the urinary output began to pick up Whether the improvement was due to the medication or to the lessening of the toxemia of the patient I am unable to say While this patient cannot as

yet be said to have recovered, she has neutralized her toxemia and survived her pneumonia She has had a crisis and now shows only the effects of her

TABLE 3

White—Female *Age thirty-two years*

Date.	Temperature	Pulse	Respiration.	Fluids, mouth.	Fluids, skin.	Total	Food, glucose	Calories	Urine c. c.
12/30	Onset of pneumonia—friction rub—bloody sputum								
12/31	103 8	124	44						
	102 8	112	38	1400	1000	2400	129	576	170
1/1	104 2	122	42						
	103 2	100	40	1560	3000	4560	290	1838	970
1/2	103 4	124	40						
	101 2	110	34	2573	1000	3573	238	1571	750
1/3	102 8	114	40						
	102 4	100	32	3230		3230	230	1599	275
1/4	101 4	104	34						
	100 6	100	30	4090		4090	328	1996	1225
1/5*	103 0	104	40						
	100 8	92	30	3695		3695	258	1417	800
1/6	101 2	120	40						
	101 0	100	36	3695		3695	222	1735	800
1/7	104 2	120	40						
	101 0	100	36	2900		2900	225	2061	1090
1/8	101 0	120	32						
	98 6	118	30	3325		3325	238	1618	1110
1/9	100 8	124	34						
	99 2	112	28	4140		4140	244	1740	725
1/10	Toxemia neutralized Afebrile Decompensation reappearing								

* New pneumonic area appeared in right middle lobe

cardiac injury Temperature ranges between 97° and 99° F Cyanosis of finger nails is absent The abdominal distention is under control

Discussion of the Treatment of Pneumonia—*Oxygen Administration*—If we are to consider the anoxemia or oxygen want, a fundamental defect which precipitates in its train so many secondary defects, it would seem rational at all hazards to provide methods and means of supplying oxygen to these patients The value of oxygen therapy has been studied and emphasized by Stadie,[11] Means,[12] and associates There can be no doubt that this measure alone is an important life saving

one There has apparently not been developed a satisfactory method of administering it so that it can be made available to the large number of unhospitalized cases needing it It is also conceivable that the agent which produces the toxemia may not be greatly influenced by the relief of the anoxemia The toxemia may be the direct result of the toxins of the bacteria Hence even when the anoxemia is relieved the major problem may still remain

Alkali Administration—Means[12] has shown graphically the value of alkalies, and the medical profession has quite generally adopted this suggestion The alkalies may be given in sufficient quantities to alkalinize the urine, and then they are to be with drawn until the urine becomes acid again

Fluid Administration—Underhill and Ringer[4] emphasized the necessity of preventing the concentration of the blood in the influenzal pneumonias They found that a preliminary venous section followed by administration of large quantities of fluids by mouth and subcutaneously prevented this concentration providing these measures were taken early enough It has long been established that fluids in large quantities are extremely important In the 3 cases cited it will be noted that the quantity of fluids averaged 3000 c c in twenty four hours

Litchfield[13] in August, 1918, emphasized the value of hyper tonic glucose, 25 per cent solutions, when given intravenously in the treatment of pneumonia Other workers have made similar suggestions The difficulty with these suggestions in the past arises from their application as sort of last measures when the patient has been allowed to slip to the brink of death The point of this clinic is not that the glucose be used as a miracle working agent, but that just as soon as the patient is taken sick it be given to him then, early, in large quantities, and by mouth If he cannot take it by mouth, give it subcutaneously in the form of 5 per cent glucose made up in doubly distilled water

I have used this method on patients late in the disease and have given hypertonic glucose solutions intravenously without affecting materially the course of the disease I believe that

this liver damage must be prevented and sidetracked if we are going to help our patients by symptomatic treatment

Drug Administration—Such drugs as are required by the patients condition should be used

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CLINIC OF DR O H ROBERTSON

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TWO CASES OF MENINGITIS DUE TO PNEUMOCOCCUS TYPE III

THESE 2 cases presented are of particular interest in relation to certain problems which arise concerning the etiology and diagnosis of pneumococcus meningitis

Case I.—P W a boy of fifteen, whose history was as follows. Four days before admission he had just returned from school for the Christmas holidays and was in good health except for a slight cold which he had had for some days On his way downtown to do some Christmas shopping he suddenly put his hand to his right forehead complaining of pain in this region This soon grew so severe that his mother took him to see the physician of the department store in which they happened to be The physician considered that the boy was probably suffering from an acute sinus infection and sent him home The pain continued and by the following day the boy seemed considerably prostrated and complained of temporal headache and superficial sensitiveness in this region Temperature at this time 104 F The family physician saw the boy and made the same diagnosis as the physician who had seen him first but on account of the presence of generalized body pains which had developed and the prostration and nausea together with high fever he considered that the boy was suffering from an attack of influenza The next morning *i e* the third day of the illness the patient seemed better His temperature was 100 5 F The headache had spread to the back of his head Sensitiveness was still present over the frontal sinus No stiffness or pain on motion of neck. That afternoon the condition became markedly worse. Temperature rose headache more severe and there was definite stiffness of neck. Mentality was quite clear and alert A consultant was called in and lumbar puncture decided on The puncture performed that evening yielded a slightly cloudy fluid apparently under normal pressure Quecksenstedt positive 32 c.c. fluid withdrawn and 30 c.c. of antimeningococcus serum given

Microscopic examination of the fluid showed 2000 cells per cubic millimeter about 60 per cent of which were polymorphonuclears Thorough examination of two stained films showed only one micro-organism an extracellular Gram positive diplococcus. The spinal fluid was cultured on blood

agar plates, in dextrose agar and in Avery's dextrose blood broth A mouse was also inoculated with a small quantity of the fluid

The patient passed a very restless night, required morphin to keep him quiet, and the following morning he was brought into the hospital in a semi comatose state By this time the organism in his spinal fluid had been identified as a pneumococcus Type III The signs were those of marked meningeal irritation, photophobia, sluggish pupils, retracted stiff neck, positive Babinski and Kernig, temperature 102° F rectal, pulse 74—good quality, respirations 24 but shallow, and the finger-nails definitely cyanotic Swelling of the right cheek and eye were noted The lungs were clear and the heart apparently normal

A lumbar puncture was performed and 30 c c of very cloudy fluid containing large flakes of fibrin were obtained The basal cistern was then punctured and an attempt made to wash out the spinal canal with physiologic salt solution After a small amount had been injected, the flow ceased and no further fluid could be either injected or withdrawn from the cistern, 25 c c of Kyes' antipneumococcus chicken serum was injected During the above procedures the patient's breathing had been getting shallower and more stertorous His condition grew steadily worse and he died fifteen minutes later

Bacteriology —The blood-agar plates showed after twelve hours a growth of large mucoid transparent spreading colonies which on staining proved to be a Gram-positive lanceolate diplococcus The Avery medium showed a heavy growth of the same organism which was bile soluble, thereby identifying it as a pneumococcus This bile autolysate added to antipneumococcus serum Types I, II, and III gave a heavy and prompt precipitate with the Type III serum The mouse which died after fifteen hours likewise yielded a Type III pneumococcus

Autopsy —An autopsy was performed within an hour after death When the calvarium was opened the dura was found to be under greatly increased tension The convolutions were flattened, the sulci less distinct, and the entire right hemisphere was covered by a greenish-yellow viscid exudate This was particularly dense in the right frontal lobe The left side was scarcely involved at all The ventricles were free from exudate The base of the cerebellum contained exudate and in the angle between the cerebellum and the medulla there was a fresh hemorrhage in the venus plexus of this point In the posterior fossa of the cranium beneath the tentorium cerebelli there was a fresh blood clot measuring 5 × 5 × 3 cm Upon opening the right frontal sinus much greenish purulent material exuded This same type of pus was found in the right supra-orbital tissues Smears made from the exudate from the right frontal sinus and from the leptomeninges showed polymorphonuclear leukocytes, mononuclear cells, and Gram-positive lanceolate-shaped diplococci The pleural cavities, lungs, and other internal organs showed no pathology Culture of heart blood at postmortem showed no growth

Case II.—W D , a man of forty, had been in good health until the present illness except for a slight coryza contracted two weeks previously Two

days before admission to the hospital pain in the left ear began rather abruptly It soon became severe and persisted for twenty four hours when the tympanic membrane ruptured with the discharge of a thin watery blood-stained fluid This brought immediate relief of the pain Twelve hours later he began to complain of frontal headache which soon became excruciating in intensity and radiated over the entire head The patient was very restless and at times delirious He was admitted to the hospital on the morning of the second day of his illness.

Physical Examination —At this time he had a temperature of 99 4° F pulse 104 and respiration 30 He was restless and uncooperative Examination of the eyes showed constricted pupils but equal and reacting to light The eye grounds revealed an early stage of neuritis of both optic nerves No paralysis of ocular muscles as far as could be determined The nose showed mucus and pus in both nostrils of a character seen in a resolving coryza The tonsils were infected and cryptic. The right ear appeared normal. The left ear was very red. The ear drum was perforated and a thin bloody discharge was seen pulsating through it There was no redness or tenderness over the mastoid nor any drooping of the canal wall Examination of the chest revealed an accentuation of the breath sounds at the right lower back where coarse râles and a to-and fro friction rub heard. Slight dulness over this area The heart appeared to be normal Blood pressure systolic 108 diastolic 90 No abnormal findings in the abdomen

Reflexes —The cranial nerves showed no disturbance except for a possible slight facial asymmetry The tongue protruded in the midline, but could not be moved very rapidly and his speech was blurred Movement of the neck was painful Abdominal and cremasteric reflexes present and equal The right knee jerk sluggish and slight the left active Ankle and plantar reflexes normal On the right the Kernig sign was positive on the left doubtful.

Lumbar puncture performed at this time yielded a yellowish cloudy fluid which exuded a drop at a time only 1 c.c. obtained A stained smear revealed large numbers of Gram positive lanceolate diplococci The cells numbered 2000 and were chiefly polymorphonuclear leukocytes

As this organism was morphologically a pneumococcus it was important to determine the type as rapidly as possible This was done by means of a precipitation test with the centrifuged spinal fluid which showed it to be a Type III Later agglutination and bile-solubility tests with a culture of the spinal fluid corroborated this determination

Following the identification of the invading organism, a second lumbar puncture was performed three hours later 20 c c. spinal fluid was obtained by using suction and 17 c.c. Kyes antipneumococcus chicken serum was given The patient was very restless and was given morphin and hyoscin which soon had a quieting effect Two hours later 40 c.c. of Kyes chicken serum was given intravenously and after a further two and a half hours another 30 c c. was given intraspinally The patient gradually became worse and died ten minutes after the last intraspinal injection

Autopsy —Examination of the brain showed that the dura mater was under considerable tension The subdural and arachnoid spaces were filled

with greenish pus which was most abundant close to the falx cerebri There appeared to be more exudate on the right side of the brain than the left and on the left the exudate was thickest over the parietal and occipital lobes The convolutions of the hemispheres were flattened The cisterns were filled with pus and there was a considerable amount of adhesion over the sylvian fissure The dura about the left lamina cribrosa was normal, but over the left tegmen tympani there was an area of discolored dura about 1 cm in diameter The cavum tympani underneath and the left mastoid cavity were full of the same thick greenish pus as was found in the meninges The right tegmen tympani was rarefied, but normal The other sinuses were normal Within the right pleural cavity there were found some adhesions at the base The right lung showed beginning hypostatic pneumonia in the lower posterior pole of the upper lobe The other organs showed the usual findings in an acute severe infection Culture from the heart's blood at postmortem yielded a pneumococcus Type III The same organism was obtained from the meningeal pus

DISCUSSION

Although pneumococcus meningitis occurs not infrequently as a complication of foci of pneumococcus infection about the brain case, the factors favoring the spread of infection to the meninges are not always apparent In the second of the above cases conditions for extension of infection seemed to be exceptionally favorable in that the tegmen tympani was of unusual thinness There was also a blood invasion, but whether prior or subsequent to the inception of the meningitis we do not know In the first case no such unusual anatomic variations were found, but the fact that an uncle of the boy had died of pneumococcus meningitis two years previously suggests the possibility that he may have had an abnormally low resistance to the pneumococcus It is probable that other factors, such as adequate drainage, etc, also play important rôles in determining the extent and direction of the spread from the original focus

Since the signs and symptoms of pneumococcus meningitis are the same as those of acute meningitis due to other pyogenic micro-organisms, the diagnosis lies entirely in the identification of the causative agent This can sometimes be accomplished very promptly after withdrawal of the spinal fluid as was done in Case II In other instances, and this is usual, the organisms are not present in large numbers and perhaps so few are visible as to make immediate diagnosis unsafe Culture of the fluid on

suitable media is then carried out It should be stressed here that pneumococci may not grow unless the media has the right hydrogen ion concentration, a p_H of 7 6 to 8, and is sufficiently enriched by fresh serum or defibrinated blood

In virtue of the fact that the spinal fluid of Case II contained such numbers of pneumococci it was possible to make an immediate typing on the cleared fluid which was rich in the pneumococcus specific soluble substance While one cannot be certain that a Gram positive diplococcus, even though lanceolate shaped, is a pneumococcus until it has been shown to be bile soluble, the presumption in Case II that the organism was a pneumococcus in view of the definite precipitation with Type III antipneumococcus serum, seemed justified as a tentative diagnosis

The results of specific treatment of pneumococcus meningitis have been decidedly discouraging While there are not a few cases in the literature of reported recoveries following the use of antipneumococcus serum, certain of these at least, as Kolmer[1] points out in his recent review of this subject, may have been due to organisms other than the pneumococcus At any rate sufficient bacteriologic data is not given to be sure of the etiology Other careful workers agree with this view[2] There are, however, a very few cases which seem to have been clearly pneumococcus meningitis and which responded to serum treatment The most favorable report on cases treated with antipneumococcus serum is that by Litchfield[3] in 1920 who employed Kyes' polyvalent antipneumococcus serum in 10 cases of pneumococcus meningitis with five recoveries The bacteriologic data given is not entirely convincing in all these cases but several of them appear to have been definitely due to pneumococcus Typing was apparently not made In view of Litchfield's report and the hopeless prognosis of the two above cases without treatment, Kyes' serum was administered The findings at autopsy showed that no form of specific treatment could have been expected to affect the disease process The abundance of exudate and deposition of fibrin were such that free circulation of immune bodies was impossible Furthermore had it been possible to administer a specific immune serum of high potency this would

have had to be given very early in the disease as the meningeal infection developed with tremendous rapidity In the second case it is probable that the meningitis began certainly not more than twenty-four hours before death

In the light of our present knowledge the greatest hope of successful treatment would seem to be in cases due to Type I pneumococcus infection since it is against this type that the most potent immune serum can be produced The most rational method of treatment consists in first washing out the spinal canal by lavage from the basal cistern through the lumbar canal, then injection of immune serum by the same route At the same time serum should be given intravenously A recent report by Harkany[4] who treated successfully a case of Type I pneumococcus meningitis with specific immune serum illustrates the possibilities of this method That there may be difficulties in carrying out this procedure was found in our cases Now that it has been possible to concentrate Type II antipneumococcus serum to a marked degree, specific treatment of meningitis due to this type may be attempted with some hope of affecting the course of the disease The use of Type III antipneumococcus serum even though increased in potency by concentration would seem to offer little prospect of influencing meningitis due to pneumococcus Type III However, the method of free subarachnoid drainage both by lumbar and cistern puncture which was employed by Globus and Kasanin[5] in a case of Group IV pneumococcus meningitis with recovery should at least be attempted When no specific therapy can be given this should be the method of choice

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CLINIC OF DRS FRED M DRENNAN AND FRANK C VALDEZ

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ESOPHAGEAL OBSTRUCTIONS

WE realize when we begin a clinic on diseases of the esophagus that we are dealing with comparatively rare conditions, but because the function of the esophagus, that of carrying food from the mouth to the stomach, is essential, we feel these conditions, though rare, are of considerable importance There may be added to this also the fact that entrance of food through other channels such as gastrostomy openings or artificially constructed tubes to replace the esophagus is painful, distressing physically and mentally, or very unsatisfactory, and a knowledge of esophageal pathology and methods of treatment which will prevent devastating surgical procedures and allow the patient to swallow his food in as nearly as possible a normal manner will seem very much worth while to both patient and physician

In considering diseases or malfunction of the esophagus, one should keep in mind the three essentials, its anatomy, its physiology, and its relation to surrounding organs The gross anatomy is comparatively simple, that of collapsible, distensible, long, narrow tube beginning opposite the upper border of the cricoid cartilage about 6 inches from the incisor teeth It is about 16 inches in length and enters the stomach about 1 inch below the diaphragm It has a caliber of about 2 cm which is slightly constricted by a sphincter at the upper extremity and by a definite sphincter at the cardia These sphincters are mentioned because a retention of irritants at either of these levels cause pathologic spasms or destruction of tissue with

scar formation The outer muscular coat of the esophagus has an external layer of longitudinal and an inner layer of circular muscle-fibers These are voluntary striped muscle-fibers near the pharynx, but below the pharynx are mostly of the unstriped involuntary type The submucosa is a comparatively thick layer and throughout the lower half has embedded in it mucous glands The mucosa is covered by a stratified epithelium which at the cardia changes to a glandular type more like that of gastric mucosa The physiology is the act of swallowing which is rather complicated, voluntary as far as it concerns the fauces and both voluntary and involuntary in the pharynx where the striped muscle is found, and involuntary below in the esophagus proper A bolus of food having been forced past the cricoid cartilage puts the muscle-fibers under tension and this starts a progressive peristaltic action which carries food downward The cardiac sphincter causes a temporary delay of food just before it enters the stomach In its anatomic relation to other organs and in its passage from the mouth to the stomach the esophagus occupies the posterior compartment of the mediastinum anterior to the loose tissue which surrounds the trachea, left bronchus, left carotid, and arch of the aorta It lies along the right side of the aorta, passing in front of it just before entering the abdomen The pleuræ are on either side of it, the pericardium in front of it, the left pneumogastric in front, and right pneumogastric behind it Posteriorly, it rests upon the vertebral column In the neck it lies very near the thyroid gland and the recurrent laryngeal nerves When one thinks then of pathology, as abscess in throat or mediastinum, glandular enlargements, heart conditions, pleural infections, aneurysms, etc, involving the esophagus directly or by pressure, it makes it necessary in one's examination and treatment to be absolutely sure of one's technic so that the patient will not be injured more than benefited by our manipulations It is recognized that rupture of the esophagus with its spread of infection into the non-resistant mediastinum is practically always fatal, so that great care is necessary in the use of tubes, bougies, esophagoscope, and even fluoroscope when esophageal pathology is present

The conditions we are considering now are those of the esophagus proper and do not include obstruction caused by pathology in neighboring organs, in other words, extra-esophageal lesions, as cervical or mediastinal glands, tuberculosis, or enlarged thyroid or thymus, mediastinal exudates, pericardial or pleural effusions, vertebral tumors of various types, aneurysms of aorta, etc. Broadly it can be said that the symptomatology of esophageal disease centers largely about the difficulty in the passage of food from the mouth to the stomach, it may be accompanied by a feeling of fulness and pressure in the mediastinal region, somewhat localized to the upper or lower levels dependent upon the location of the lesion, seldom definite or severe pain unless perforation or near perforation has taken place, regurgitation of food and secretion (not a real act of vomiting) depending somewhat on the tightness of the obstruction, retention of food particles for hours in certain types of lesions, loss of weight and strength, part or all of which may be explained on the basis of insufficient food intake, thirst, dryness of skin, and diminished urinary output from insufficient fluid intake, and in all malignancies, blood in bowel movements, secondary anemia, and mild leukocytosis

Lesions of the esophagus may be classified in the usual way of (1) congenital, (2) acquired

CONGENITAL LESIONS

Congenital lesions are considered first, not because of their frequency or importance, as they are rare, but because in teaching it is sometimes better to get the less important covered first so that your attention will remain fixed for a longer time on the more important conditions Congenital lesions vary greatly from small areas of partial obstruction due to deformity of the wall, or to folds of mucous membrane overgrowth, to complete obstruction with parts of the esophagus represented by fibrous cords in which there is no indication of a lumen This cord like structure may connect with a pouch above or below or the pouches may connect with bronchi or trachea or end blindly In these cases of total obstruction diagnosis is usually not made.

the babies may show some other congenital deformity, and do not survive The partial obstructions are more amenable to treatment

Case I.—Our first case is that of a boy who came to us when three years of age His complaint was that of regurgitation of food, a condition which had been present since birth The regurgitation would begin as soon as food was taken, it was worse with solids than liquid foods, though everything caused trouble Careful diet as prescribed by pediatricians had failed to help He was markedly emaciated, showed all the signs and symptoms that go with under nourishment There was no history of swallowing foreign body or escharotics and his symptoms began in the first weeks of life before it would be logical to expect a baby to have placed foreign bodies in his mouth Wassermann was negative Bougie and tube were obstructed at about the level of bifurcation of trachea and barium was held at same level, the lower edge of the barium shadow being smooth and regular There was no blood in the bowel movements, no pain on swallowing, in fact the child seemed to be hungry and thirsty enough so he wanted to take food whenever it was offered, speaking against ulcerations with inflammatory reactions This definitely is a congenital deformity which did not completely obstruct

The method of dilating used was that developed by Dr Bertram W Sippy It consists of having the patient swallow a small silk thread which has been waxed to prevent untwisting and knotting Usually it is necessary to have at least three yards swallowed so that the lower end is well down in the small intestine and consequently so fixed that it cannot be pulled out We have found by experience that this thin Corticella silk thread will pass through any opening that will allow water to trickle through so that even the patients with very tight obstructions are able to get it through if they perservere in the attempt The string is used as a guide and along it the wire is inserted The wire is of flexible steel with a small bulb screwed and soldered to the small end This bulb has an opening in its center through which the string is threaded The dilating bulbs are arranged in sequence on the wire, a small one first, gradually increasing in size so that the center bulb does the dilating A similar series turned in the opposite direction allows for a safe and harmless removal of the dilating outfit Usually five or seven bulbs make up the series The bulbs are then pressed along the wire through the obstruction by a spiral The entire outfit, wire, bulbs, and

spiral, are all flexible and can be used through tortuous obstruc tions as readily as through straight strictures The safety of this method lies in the fact that the string has to follow the opening, the wire of necessity must follow the string through the opening and the dilating bulbs must follow the wire, thus assuring us against false passage and giving a maximum lateral dilatation with a minimum danger of perforation This boy has

Fig 61 —Congenital obstruction (partial) (Courtesy of the Illinois State Medical Journal.)

been dilated to date, has had a normal development, and has very little difficulty in getting ordinary foods through During the past few years only occasional dilatations have been necessary, more as a matter of being sure that the opening was not getting smaller, than for real difficulty in swallowing (Fig 61)

In such cases we cannot be sure of type of tissue involved in the obstruction or the length of the deformed area because the

barium shadow below the obstruction is likely to be the size of the opening at the tightest point of obstruction or smaller, certainly not large enough to be the outline of a normal esophagus, although the esophagus below the point of obstruction may be a normal size The dilatations in this case are practically always accompanied by slight bleeding indicating some tearing of tissue This almost surely has led to some scar tissue formation

ACQUIRED LESIONS

Acquired lesions, if we make our list complete, should include

1 Inflammations caused by chemical or mechanical agents plus infections with resulting reaction and scar tissue formation

2 Some special inflammations as tuberculosis, typhoid, syphilis, thrush, etc , uremic ulcers, occasional peptic ulcers, etc

3 Diverticula which are divided into three types depending on their location and type of development

(*a*) Traction diverticula which may occur in any portion of the esophagus but are more frequently found in the lower portion They are seemingly caused by the wall of the esophagus being involved in inflammatory reaction in regional glands, mediastinal tissue, etc When the inflammatory reaction quiets down, scar tissue develops, and since the tendency of scar tissue is to contract, the wall of the esophagus is deformed by the scar tissue contraction and a diverticulum results This type usually does not cause symptoms and is found during careful fluoroscopic examination where the esophagus is examined during the ingestion of the opaque barium meal or at autopsy Since there are no difficulties caused by this type, no treatment is necessary or advisable

(*b*) Pressure diverticula are usually found on the posterior wall at the upper end of the esophagus at its junction with the pharynx where the muscle coats do not completely surround the tube It is held that the act of swallowing causes pressure against this point of weakened musculature and a portion of the thin inner wall of the esophagus is crowded between muscle-fibers, giving a pouching diverticulum With a pouch formed, each peristaltic wave gives more pressure, in time food and secretion

enter the pouch, tending to distend it more and more, till finally it is large enough to give symptoms as in the next case

Case II.—Our second case came to us with a history of difficulty in swallowing which had been present for a year gradually getting worse with milder symptoms for a long time which were so slight that they were not considered of any importance. He noted a sensation of food sticking high up in the esophagus the sensation getting worse as he continued to eat until there would be a regurgitation of food with a peculiar crowing sound He had brought up food in this way that had been eaten twenty four hours before The condition was made worse by excitement He had lost 40 pounds in weight and was definitely weak seemingly because of a lessened food intake

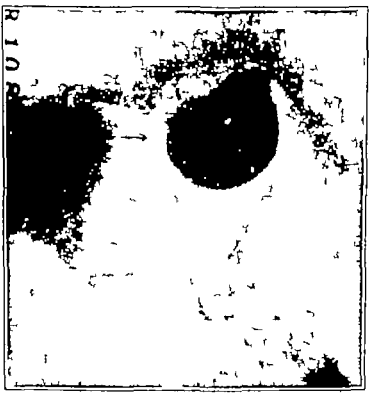

Fig 62.—Esophageal diverticulum. (Courtesy of the *Illinois State Medical Journal*)

No tumor masses or glands were noted in the neck. Wassermann was negative Stools were negative to blood Physical examination was essentially negative except for difficulty in swallowing a definite fulness above the left clavicle to left of midline on swallowing and a regurgitation of food quickly after its ingestion with the peculiar crowing sound noted The patient was x rayed early in examination because the type of difficulty the crowing sound on regurgitation the pouching above clavicle and the retention of food spoke for a diverticulum. At fluoroscopic examination we found the large pouch as seen in Fig 62 under the fluoroscope the stomach tube and bougies could be passed through the esophagus into the stomach This was done under direct observation because if the opening into the pouch had been large

enough to allow entrance of the tube, slight pressure might rupture the sac and cause serious infection The patient then swallowed the silk thread, as described in first case, and large bulbs were passed proving that the difficulty in swallowing was not due to anatomic narrowing of the lumen, but to pressure obstruction by the filled pouch Diverticuli of small size are usually causing so little trouble that operative procedure is not justified, but this man was having so much trouble that operation was advised

There is always a tendency for the surgeon to feel that radical removal should be done because of the ease with which the sac can be isolated and perhaps the anatomic comparison to an ordinary hernial sac, but experience has shown that removing a diverticulum will be followed fairly regularly by leakage,

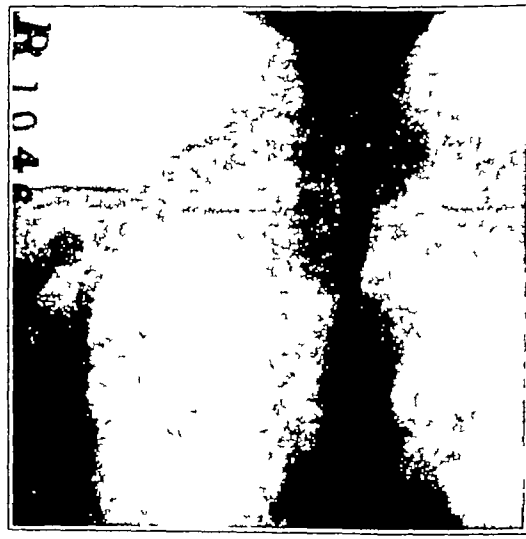

Fig 63 —Diverticulum after operation (Courtesy of Illinois State Medical Journal)

mediastinitis, and death, so more conservative measures are advisable, such as two-step operations with the sac exposed till adhesions are formed and then removed, or the invagination operation as sponsored by Dr Sippy and Dr Bevan, care being taken to fix the invaginated mass so that it will not be forced into the esophagus and interfere with respiration or cause an obstruction during convalescence

The invagination method was used in this case, Dr Bevan doing the operation The patient made an uneventful recovery and has had no difficulty in swallowing since Figure 63 shows present condition of esophagus, a little retention but not causing any trouble

Case III.—Diverticulum high up in esophagus small in size causing only a regurgitation of food but no pain and not being inconvenient enough to justify operation with its attendant dangers (Fig 64)

Fig 64.—Diverticulum at upper end of esophagus (Courtesy of Illinois State Medical Journal)

Case IV.—A traction diverticulum at the lower portion of esophagus found in a patient who came to us with a duodenal ulcer This diverticulum was not causing any symptoms and was found only in routine x ray examination This is probably a result of contraction of healing mediastinal glands which were attached to the esophagus wall (Fig 65)

(*c*) A combination of the above two types or a pressure-traction type This type may be seen at any portion of the esophagus where a weakness of the wall has been caused by inflammatory reaction, scar tissue, etc The essential point is that in the development of the traction diverticulum from scar

tissue contraction, the tip of the pouch is below the opening and the opening is large enough and so located that food and secretion are crowded into the pouch by the peristaltic action of the muscular coats during the act of swallowing If located

Fig 65 —Diverticulum in lower portion of esophagus (Courtesy of Illinois State Medical Journal)

anywhere except at the upper extremity of the esophagus, surgery is not logical

4 Cardiospasms, usually at the lower end of the esophagus, occasionally at the upper end Perhaps this can best be treated by taking the history of the next case to be demonstrated

Case V —A young man fairly well nourished, giving a history of difficulty in swallowing for fifteen years, coming in attacks during which he would have a great deal of difficulty, with periods of freedom during which time he could swallow all sorts of food and drink normally, gradually increasing trouble, so that at the present time he has difficulty at every meal At times he will have difficulty at the beginning of the meal, regurgitate food that is being held in the esophagus, and then be able to eat the rest of a normal meal without difficulty He has no real pain, just a sense of fulness and pressure at the lower portion of the esophagus The regurgitated food is undigested, it does not taste sour or bitter, and occasionally contains food that has been eaten twenty-four hours previously It has no blood in it, but is intimately

mixed with strands of thick tenacious mucus The patient has noted food particles in mouth on cheek, and on pillow in the morning after a comfortable night s sleep Cold foods cause more difficulty than hot and liquids more than solid foods nervousness excitement and anger always make the condition worse

Fluoroscopic examination as seen in Fig 66 shows a typical cardiospasm picture a marked retention of barium the dilatation of the esophagus the presence of visible peristaltic waves indicating a hypertrophied muscle barium

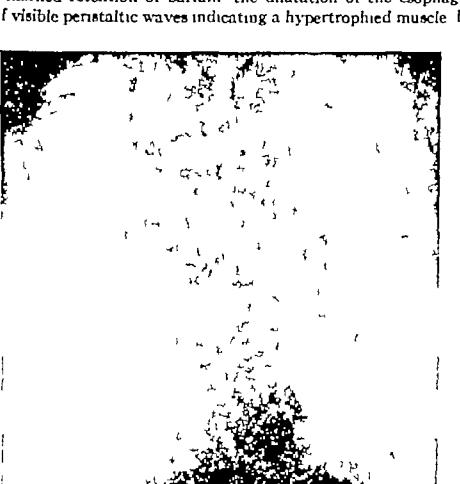

Fig 66 —Cardiospasm

passing through in clumps not the thin stream characteristic of an anatomic obstruction and the smooth regular cone shaped lower border to the shadow The diagnosis of cardiospasm is so easy from the clinical history and *x* ray examination that we are prone to forget or fail to do the ordinary clinical tests which are more accurate and safe than *x* ray They are based on the fact that a cardiospasm is the only type of obstruction which is tight enough to hold water but at times will allow the passage of food and instruments with

only slight pressure The patient is allowed to drink cold water till he feels his esophagus is completely filled and taking more would cause regurgitation. Then as gently as possible an ordinary Ewald stomach-tube is passed The suction bulb is attached as soon as the tube enters the upper end of the esophagus and as the tube is lowered the content of the esophagus is aspirated Amount aspirated is found to be 230 c c (about 2⅓ times the amount held by the sphincter of a normal esophagus—100 c c) This material has a great deal of stringy mucus in it and with dimethyl-amido-ozo-benzol as a test, shows an alkaline reaction and contains no food Then by gradual increased pressure the tube can be felt to pass into the stomach, indicating that the obstruction which was tight enough to hold water has allowed the passage of an ordinary Ewald tube without pain The suction bulb is again applied and content of aspirated material noted, now it contains food material and with the dimethyl gives a definite reaction for free hydrochloric acid indicating without doubt that the tube is now in the stomach With the tube still in place, a deep blue solution of methylene blue is pumped into the stomach through the tube, then enough clear water so that the tube is washed clean of the blue as it is removed from the stomach If conditions are right, we should have the methylene-blue solution in the stomach and the esophagus empty Patient is now asked to again drink cold water until the esophagus seems full and as before the stomach-tube is carefully inserted (care being used so patient will not vomit due to the irritation of the tube) The suction bulb is attached as the tube enters and approximately the same amount (230 c c) of clear water is removed from the esophagus, slight pressure again is used, the tube is felt to slip through into the stomach and the methylene-blue solution is aspirated This test can be done anywhere without the use of elaborate or expensive equipment, is safe in its technic and less likely to error even than the interpretation of *x*-ray pictures Clinically it is also good mental training to have methods of diagnosis that use our heads and hands, do not require *x*-ray or some other of our instruments of precision upon which we have a tendency to lean too much

The treatment here varies only in the type of dilating apparatus The patient swallows the silk thread as described in Case I The wire is passed along the thread and large bulbs passed along the wire through the spasm into the stomach, again proving it to be not an anatomic obstruction This extra precaution, we feel, is necessary because if the cardiospasm dilator were used in an anatomic stricture rupture would almost surely result This dilator consists of a bag, approximately 6 inches in length and in this case 5 inches in circumference, which passed along the wire until its center was at the cardiospasm The bag is connected by a tube with a pressure bulb and mercury manometer The position of the bag can be checked

by measurements or by fluoroscope, and when it is found to be in position it is dilated by forcing air into it with the bulb, the amount of pressure being registered by the manometer The attempt is made to dilate so that there will be a slight tearing of the muscle fibers, and if this is accomplished, there will be a slight streak of fresh red blood on the apparatus when it is removed It is well to distend the bag after it is removed so that we can be sure it is working properly and that under pressure it is dilated to a definite size It is possible in any given case that the size of the bag may have to be increased, but when the proper dilatation is accomplished patients get definite and almost immediate relief

5 Obstructions, scar tissue from a variety of escharotics chemical, mechanical, and thermal

Case VI.—A history of swallowing lye when a child Severe acute pain spasm at once and inability to take food Gastrostomy was done almost immediately and no dilatation for four or five years The obstruction was

Fig 67.—Cicatricial obstruction (Courtesy of Illinois State Medical Journal)

complete enough so that at no time could any barium be seen to trickle through the esophagus A string could not be swallowed Esophagoscopic examination could not locate the opening so dilatation was not attempted (Fig 67)

While we were not able to get a string through in this particular case, this is the type of condition when the Sippy dilating bulbs give the best results, by using small bulbs first and increasing the size slowly, the tightest type of scar tissue can be stretched out comparatively safely and it will remain open very satisfactorily

6 Tumors, usually carcinoma, occasionally sarcoma

Case VII.—A man, fifty-six years old, came to us with a history of difficulty in swallowing, present for four months, first noted with solid foods, but gradually getting worse, so that at the time of first examination he could get only small amounts of water through He was emaciated, showed occult

Fig 68.—Carcinoma (Courtesy of Illinois State Medical Journal)

blood in every bowel movement, had a secondary type of anemia, and negative Wassermann Bougie met with obstruction 14 inches from the incisor teeth and x-ray showed barium retention with the lower edge of barium shadow being markedly irregular as seen in Fig 68 This is the location of about 50 per cent of esophageal cancer

Case VIII.—A man of fifty, who had difficulty in swallowing for three months, beginning with choking on a piece of meat, but at the time of first examination was able to get only small amounts of water through He had lost weight and strength rapidly, was very much dehydrated, Wassermann negative, blood present in bowel movement x-Ray examination showed

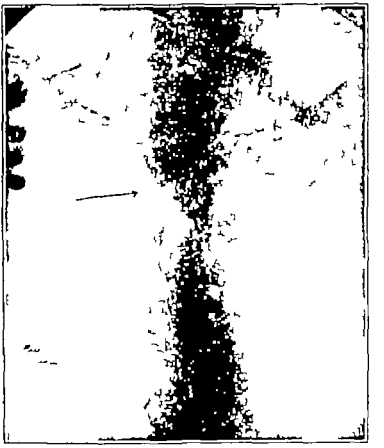

Fig 69.—Carcinoma (Courtesy of the Illinois State Medical Journal.)

Fig 70.—Carcinoma (lateral view) (Courtesy of the Illinois State Medical Journal.)

obstruction (Figs 69, 70) located at about the bifurcation of the trachea, the next common location for esophageal cancer (40 per cent)

Case IX—A woman, forty-eight years old, who had been having difficulty in swallowing for two months so severe that she felt she was starving to death Bougie met with obstruction 15 inches from the incisor teeth, which we felt should be at the lower end of the esophagus She also had negative Wassermann, positive blood in bowel movements, and had lost rapidly in weight and strength She had a definite epigastric tenderness and rigidity Though a mass could not be palpated, x-ray revealed an infiltrating mass along the lesser curvature of the stomach involving the esophagus and causing the obstruction

The malignancy cases are dilated as described in the first case, care being particularly necessary not to attempt too rapid dilatation, as friable cancerous tissue is easily torn and rupture of the esophagus in such cases is always serious We are usually able to get a large enough opening so that starvation or dehydration is prevented

The 3 cases were treated by the method described for the congenital obstruction In each of the cases the string was swallowed with some difficulty, but after it was down dilatation was accomplished and a large enough opening made so that liquids could be swallowed readily, semisolid foods easily, and some solid foods without great difficulty

Surgery of the esophagus is very difficult and unsatisfactory Radium in malignancy is of questionable value, deep therapy in scar tissue and in our congenital obstructions of no value Since the function of the esophagus is to allow the passage of food and water, we have felt that the wire and dilating outfit designed by Dr Sippy furnishes the best method of treating these cases To recapitulate briefly, the method requires the patient to swallow a silk thread, waxed to prevent kinks and knots, until it becomes fixed in the intestine This is used as a guide for the wire and insures us against false passage as no other method does A gradual wedge of bulbs is then placed on the wire, both for dilatation as the wedge enters and for safety as it is withdrawn This wire makes the use of a considerable pressure possible The graduated bulbs make a lateral dilatation with-

out tearing The flexibility of the outfit makes it possible to follow a tortuous path safely and withal gives us the maximum amount of dilatation with a minimum amount of danger, and makes it possible in our first case to keep him living a normal life The same in scar tissue cases and in neoplasms, to keep them happy, fairly comfortable, and able to swallow food and fluid until metastases have caused constitutional symptoms

CLINIC OF DR NATHAN S DAVIS, III

HYPERTENSION CLINIC OF NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

FOUR CASES OF ARTERIAL HYPERTENSION WITH ELECTROCARDIOGRAPHIC STUDIES

Case I.—E. H., a white woman, aged sixty-three years, 65½ inches tall and weighing 148 pounds, was admitted to the Medical Clinic December 21, 1926, complaining of swelling of feet in the evening, occasional swelling of face and hands, rheumatism, vertigo, deafness, and rather indefinite gastro-intestinal symptoms with belching and some nausea and vomiting as a rule during the night and not related to meals, all of which symptoms had been present off and on for several years. The rheumatic trouble had been present more or less since she had an attack of inflammatory rheumatism in 1906. She stated that in September, 1926, she had a stroke, during which she did not lose consciousness, but which caused her to be confined to her bed for several days, during two of which she had loss of control of the urethral and anal sphincters. She had occasional nosebleeds and regularly had to get up at night to urinate.

She had had measles, chickenpox, and scarlet fever in childhood, jaundice when four years old, inflammatory rheumatism at forty-three, in 1906, and influenza in 1918. Her menstrual history was normal; no pregnancies; menopause at fifty-thirteen years before admission.

Her father died at sixty-five with edema, heart or kidney disease; her mother at eighty-one of pneumonia; one sister died in 1923 after having had three strokes.

Physical Examination.—The eyes react to light; the fundi are normal; several carious teeth with alveolar abscess; chronic tonsillitis; nerve deafness. Heart and lungs normal; on a 2 M. plate the transverse diameter of the chest is 25.2 cm.; the right border is 4.7 cm. and the left 9.5 cm. from the midline. Abdomen negative except slight tenderness over splenic flexure of the colon. Table 1 (page 400) gives a summary of the blood pressure, laboratory findings and the therapy employed.

In January, 1927, she had six teeth extracted, following which she felt better except that shortly after this she had a carbuncle on the scalp in the occipital region which was treated surgically. In February, 1927, the gastro-intestinal symptoms and headache were more severe and she had a roentgenologic examination which revealed a pylorospasm, but with no evidence of ulceration. An Ewald test breakfast showed a total acidity of 63 and a free

TABLE 1

Date	Blood-pressure	Therapy
12/21/26	200/100	Ca lact 4 t i d
12/28/26	178/90	Ibid
1/ 4/27	172/86	Ibid
1/11/27	172/90	Ibid
1/18/27	176/96	Ibid
1/25/27	176/90	Ibid
1/31/27	200/110	Ibid
2/ 8/27	154/86	Ibid
3/ 1/27	168/90	Ibid
3/22/27	140/90	Ibid
5/10/27	146/90	Ibid
6/ 1/27	200/145	Ibid
7/20/27	180/90	KI 0 6 and $CaCl_2$ 0 6 t i d
8/ 3/27	160/90	Ca lact 4 t i d
8/17/27	170/86	Ibid
8/31/27	152/80	Ibid
9/14/27	190/110	Ibid
9/28/27	145/100	Ibid
10/14/27	190/100	(Furuncle on left cheek) Ca lact 4 t i d
10/19/27	170/90	Ca lact 4 t i d
11/ 2/27	150/80	Ibid
11/23/27	180/100	Ca lact 4 t i d and KBr 0 9 t i d
12/14/27	150/100	Ibid
1/ 4/28	170/98	Ibid
2/ 1/28	195/110	Ibid
7/25/28	180/115	Bi Subnit 0 3 t i d
8/15/28	164/88	Ibid
9/ 5/28	156/86	Ca lact 0 4 t i d

Urine

Date	Albumin.	Sugar	Casts	Specific gravity	Phenolsulphonephthalein 15 min	30 min	60 min	Total
12/21/26	Trace	0	0	1019				
1/ 3/27	0	0	0	1020				
1/25/27	0	0	0	1016				
3/11/27	0	0	0	1020				
7/21/27	0	0	0	1017	20%	15%	15%	50%
7/25/27	Trace	0	0					

Blood

Date	Red cells	White cells	Hemoglobin	N.P N	Creatin	Sugar
1/ 3/26	4,380,000	20,600	90			
12/24/26				54 5	1 2	102
3/11/27		8,050				

hydrochloric acid of 50, no blood or other abnormal constituents Her condition has remained about the same since this time though she had a furuncle on the face in July, 1927 and furunculosis in both axillæ and left groin which came on after she had been bitten by bedbugs in July, 1928 She has been able to work most of the time as an office assistant in a sanitarium or as a house- or nurse-maid When she has been at work the blood-pressure has been slightly higher than when she has been resting

This is an average case of vascular hypertension of several years' duration in a woman much more intelligent than the average clinic patient As a

result it is probable that she has followed the diet (low in salt and protein except milk protein) prescribed and so diet has possibly had more to do with the improvement shown than in the cases to follow There was no anemia the urinary and blood findings reveal but slight renal damage and the *x* ray and electrocardiogram (Fig 70*a*) show no significant changes either in the

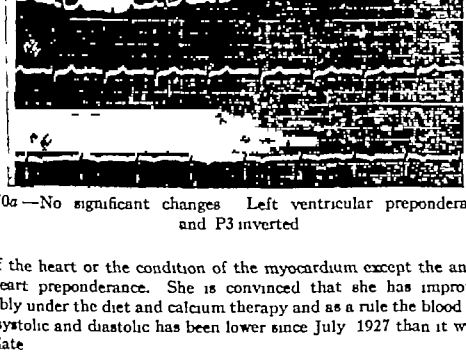

Fig 70*a*—No significant changes Left ventricular preponderance T3 and P3 inverted

size of the heart or the condition of the myocardium except the anticipated left heart preponderance. She is convinced that she has improved considerably under the diet and calcium therapy and as a rule the blood pressure both systolic and diastolic has been lower since July 1927 than it was before that date

Case II.—S A. a colored woman aged fifty two years 64½ inches in height and weighing 146½ pounds was admitted November 26 1927 complaining of pains in the joints of four years duration heavy feeling in the stomach after eating intermittent nocturia The joints of the extremities especially those of the hands ankles and elbows were painful and one joint after another had become involved The feeling of heaviness in the epigastrium was associated with belching There had been some paroxysms of precordial pain which did not radiate and were not associated with food taking during about three years Two weeks before admission she had an attack of vertigo lasting one half hour There was slight dyspnea on exertion

She had had measles in childhood and influenza in 1918 Menopause in 1919 She had had seven children three of whom died in childhood the youngest was born in 1922 Husband has tuberculosis Mother died of rheumatism father of unknown cause.

Physical Examination.—Fundus examination shows in both macular regions but especially in right one extensive areas of choroidal pigment atrophy which have a tendency to assume a stellate arrangement and through which the grayish white sclera shows. Many smaller areas of atrophy between disks and macula Both disks have a somewhat waxy appearance and the

retinal arteries are somewhat narrowed Fairly deep physiologic cupping Bilateral central choroiditis and retinal arteriosclerosis, eyes react to light Carious teeth, lungs negative, abdomen negative Left heart dulness 12 5 cm to left, right 3 cm. to right of midline, there was a systolic murmur at the apex and an accentuated second sound especially in the aortic area Reflexes normal

A summary of the laboratory findings, blood-pressure, and therapy is shown in Table 2

TABLE 2

Date.	Blood pressure	Therapy
11/29/27	210/152	
12/ 6/27	252/140	Ca lact , lactose āā 2 q i d
1/ 3/28	220/150	Ibid
1/ 4/28	168/118	Ca lact 4 t i d
1/11/28	248/140	Ibid 8 t i d
1/25/28	264/150	KBr, $CaCl_2$, āā 0 6, NH_4Cl 0 3 t i d
2/ 1/28	170/90	$CaCl_2$ 4 t i d
2/15/28	280/160	KBr, $CaCl_2$, āā 0 6, NH_4Cl 0 3 t i d
2/29/28		Ibid
3/14/28	246/130	Ibid
3/28/28	260/135	Ibid
4/11/28	248/130	Ibid
4/25/28	260/152	Ibid
5/ 2/28	245/130	Ibid + $CaCl_2$ 8 t i d
5/23/28	224/128	Ibid
6/ 6/28	250/150	KBr, 0 9, $CaCl_2$ 0 6 t i d
6/27/28	270/150	KBr, $CaCl_2$ āā 0 9 t i d
7/11/28	230/133	Ibid
8/ 8/28	212/119	Ibid
8/29/28	220/120	Ibid
9/12/28	230/150	Ibid

Urine

Date.	Albumin.	Sugar	Casts	Specific gravity	Phenolsulphonephthalein 15 min	30 min	60 min	Total
11/26/27	+	0	hyal	1005				
1/11/28	+	0	gran	1015	12%	18%	18%	48%

Blood

Date.	Red cells.	White cells	Hemoglobin	N.P.N
11/29/27				44 2
1/ 4/28	4,290,000	5250	80	

Up to the present time her condition has remained about the same, though subjectively she is feeling much better than on admission This is a much more advanced and severe case than the first one There is much vascular change in the retinæ, there is considerable cardiac enlargement, and the electrocardiogram (Fig 71, *b*) with its inverted T in all leads and long slurred QRS is indicative of serious myocardial damage The specific gravity of the urine is lower, and albumin and casts have been found at all examinations, though the phenolsulphonephthalein excretion was 48 per cent She has been unable to follow the prescribed diet and objectively the calcium therapy has had little effect but subjectively her condition is greatly improved and she feels so well that she does not visit the clinic as often as directed

As to prognosis, this is a case in which death due to cerebral accident

is to be expected though heart failure or coronary occlusion or acute infectious disease must not be excluded Uremia will probably not be a complication

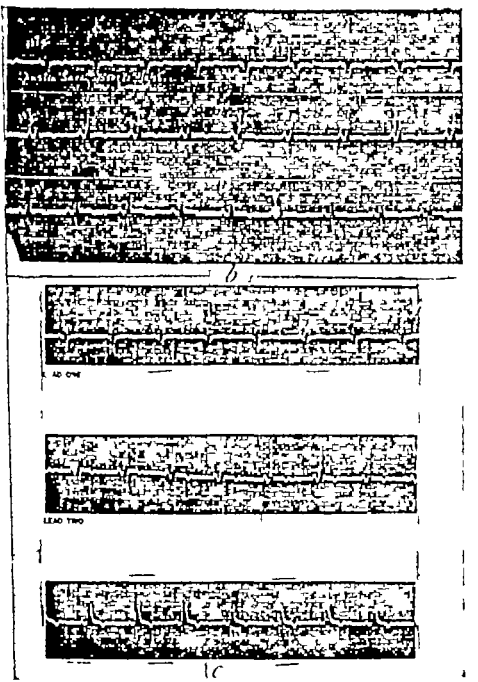

Fig 71 —*b* QRS 0 1 sec. slurred in all leads T inverted in all leads
c Same as *b* except that T1 is upright

Case III.—G F a negro male aged seventy five years was admitted to the Medical Clinic July 15 1924 complaining of hiccough vomiting pain in shoulder and upper arm He had had hiccough off and on for fifteen years but no vomiting till day before admission. He had vertigo palpitation and some dyspnea on exertion nocturia constipation and had had whooping-cough measles, rheumatism, and malaria. He denied venereal infection Family history was negative.

Physical Examination —Pupils reacted to light and accommodation

there was an arcus senilis and a chronic conjunctivitis Heart and lungs were negative Abdomen was negative Reflexes normal Wassermann negative Laboratory findings, blood-pressure, and therapy are shown in Table 3

TABLE 3

Date	Blood pressure.	Therapy
7/15/24	180/110	$NaNO_2$ 0 05 q3h, (hiccough)
7/22/24	140/100	
9/26/24	135/80	
8/27/26	175/100	Nitroglycerin 0 0006 q2–4h for pain
3/18/27	218/140	KBr 0 78, $CaCl_2$ 0 78 t 1 d
3/23/27	178/130	
3/30/27	180/120	
5/ 4/27	170/100	
5/15/27	180/110	
5/25/27	190/100	KBr 0 6, $CaCl_2$ 0 6 t 1 d
6/ 8/27	190/150	Ca lact 4 t 1 d, Tr digitalis 2 t 1 d
6/22/27	154/100	NH_4Cl 0 3, $CaCl_2$ 0 45 t 1 d
7/ 6/27	160/110	NH_4Cl, $CaCl_2$, KBr āā 0 3 q 1 d
8/ 3/27	160/110	KI, NH_4Cl, $CaCl_2$ āā 0 3 t i d
8/10/27	170/110	Ibid
8/24/27	150/100	Ibid, nitroglycerin 0 0006 q 1 d
10/12/27	154/100	KBr, KI āā 0 6, $CaCl_2$ 0 3 t 1 d
10/26/27		Ca lact 4 t 1 d
11/ 9/27	150/100	NH_4Cl, KI āā 0 6, $CaCl_2$ 0 38
11/30/27	175/100	Ca lact 4 t 1 d
1/11/28	190/100	Ibid Dig fol 0 06 b 1 d
1/25/28	210/130	Ibid
2/15/28	180/90	Bi subnt 0 3 t 1 d, digitalis fol 0 06 t 1 d
2/29/28	210/110	Ca lact 4 t 1 d
3/14/28	180/110	Ibid
3/28/28	200/110	(Coryza) Bi subnitrate 0 3 t 1 d , dig fol 0 06 t 1 d
4/18/28	170/110	Ibid
6/13/28	170/120	No medicine for five days before
6/20/28	170/80	Ibid
8/ 8/28	170/106	No medicine for past week Ibid
8/29/28	190/100	$CaCl_2$ 0 36 t 1 d
9/12/28	180/120	Ibid

Urine

Date	Albumin	Sugar	Casts	Specific gravity	Phenolsulphonephthalein 15 min	30 min.	60 min.	Total
5/ 3/27	Trace	0	0		9%	18%	18 5%	45 5%
3/15/27	"	0	0					
6/ 8/26	0	0	0	1018				
5/ 8/26	++	0	hyal ++					
8/25/24	0	0	0	1009				
8/ 5/24	0	0	0	1014				
7/15/24	+	0	hyal and epithel	1020				
4/27/27	0	0	0					
4/12/27	+	0	hyal and gran ++	1015				

Blood

Date.	Red cells.	White cells.	Hemoglobin
10/24/24	5,006,000	6040	90
3/14/28	5,480,000	7500	85
4/19/27	Guanidin 0 48		

The hiccough persisted for five days and then stopped to return again a few days later Benzyl benzoate miscible 0 9 c c was prescribed and the

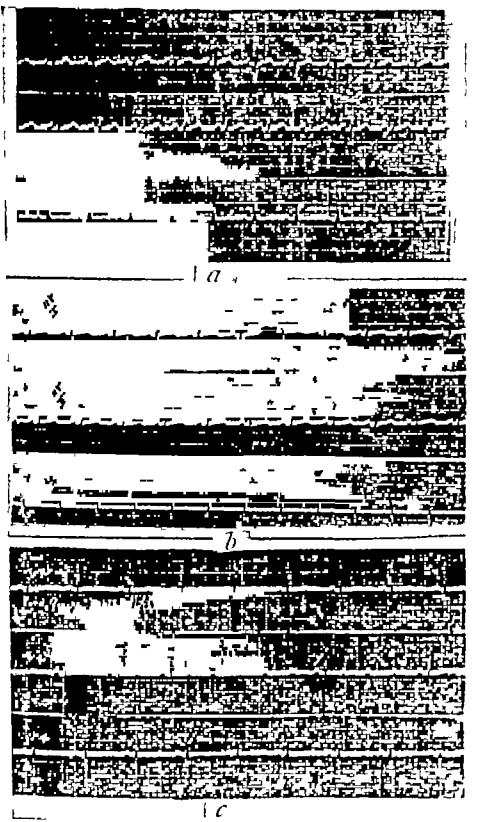

Fig 72.—*a* 3/23/27 Normal mechanism.
b 5/4/27 T1 diplasic left ventricular preponderance
c 1/11/28 P1 2 3 notched and broad QRS slurred in all leads

hiccough ceased, only to recur in about one month or six weeks A gastro intestinal x-ray examination on October 10, 1924 was entirely negative Potassium iodid was prescribed in 0 9 gm doses three times daily and from that time on (September 26, 1924) there has been no hiccough On January 13, 1925 he complained of pain over left chest and above left eye which lasted for a week or two and was relieved by iodids and salicylates

On May 18, 1926 he returned complaining of cough, weakness, and insomnia, and on June 8, 1926 of precordial pain which was referred to the back but was not typical of angina pectoris There was no evidence of aneurysm or pleurisy In August this pain was present every evening, and was worse when he was tired or after exertion Nitroglycerin 0 0006 gm gave relief, and he did not return until March 18, 1927 when he complained of dyspnea on exertion, a feeling of weight in the precordium, and pain radiating to the left shoulder and arm, and a non-productive cough He was given nitroglycerin 0 0006 as necessary, potassium bromid 0 38 and calcium chlorid 0 38 in simple elixir three times daily, and was put on a low maintenance protein, low salt diet Under this management he improved somewhat and did not complain of precordial distress In January, 1928 had a cold and with it a severe cough and considerable dyspnea on exertion which was relieved by digitalis and calcium In March, 1928 he had another cold and with it a feeling of weakness and some precordial pain He continued in good condition after this until October when he developed a metatarsalgia and on October 24th complained of orthopnea

This man has been under observation for four years At the beginning his most marked symptom was hiccough which has not been present since 1924 Since 1926 an anginal pain has been the chief cause for complaint, and electrocardiograms (Fig 72 *c*) taken at intervals since March, 1927, when it was normal, have shown increasing evidence of myocardial damage He is not anemic and the urinary findings have not changed materially His blood guanidin, according to the method of R H Major, is distinctly elevated During the last year or so his pressure has averaged higher than before despite the calcium therapy which has been used for the most part and the bismuth subnitrate which has been tried a little It is probable that this man will some day have a coronary occlusion that will cause death, though a cerebral accident or acute infectious disease may intervene Uremic symptoms, with which death is usually associated in the younger individuals with the more malignant type of the disease, are most unusual in the more chronic forms These remarks apply also to the next case

Case IV—M S, No 45767, a colored woman, aged fifty six years, was admitted to the Northwestern University Clinic April 29, 1919, stating that she had had a paralytic stroke in 1917, two years before admission, and one in April, 1919, six months before admission, which involved the right side and caused blindness for several days At the time of admission she was complaining of severe pain in her arms and legs, headaches, and nocturia At the first admission there were no notes as to the blood-pressure, physical findings, previous illnesses, etc, but on December 21, 1919 it was noted that she had a negative Wassermann reaction in the blood, that the blood

pressure was 190/115 that there was a systolic murmur at the apex and that the aortic second sound was markedly accentuated On October 12 1920 her pressure was 220/130 and she was bled 360 c c. being removed (For further blood pressure readings and notes on medication see Table 4)

TABLE 4

Date.	Blood-pressure.	Therapy
10/21/19	119/115	
10/12/20	220/130	
5/ 9/21	210/110	
6/20/21	210/128	
10/27/21	180/100	
12/11/21	190/100	
3/30/22	200/104	
8/24/22	202/104	
3/12/23	180/110	
4/ 5/23	198/100	
7/ 5/23	220/110	(Rest in bed)
7/ 9/23	160/90	
3/11/24	200/120	Ca lact 8 t. i d
3/25/24	170/100	Ibid
12/10/23	220/100	
12/27/23	198/110	
2/14/24	210/100	
3/ 6/24	180/94	
6/ 8/25	174/98	
6/18/25	168/90	Ca lact 8 t. i d
11/ 5/25	182/106	Nitroglycerin 0 0008 b i d
11/20/25	160/94	Ca lact 0 6 t i. d
1/ 5/26	182/100	$CaCl_2$ 0.38 t. i d
1/15/26	190/110	KCl 0.38 $CaCl_2$ 0 76 t i d
2/16/26	164/100	Ibid Nitroglycerin 0 0006 t i d
3/ 9/26	168/100	Ibid
3/23/26	170/100	$CaCl_2$ KCl 0.3 t i. d
4/23/26	190/100	
6/ 8/26	198/100	$CaCl_2$ 0.3 q i d
8/13/26	190/130	Ibid
8/20/26	150/110	Ibid
9/10/26	180/120	KI NH_4Br $CaCl_2$ āā 0 3 t. i. d
9/24/26	186/112	Ca lact Lact āā 8 t. i d
10/22/26	190/120	Lact 16 t. i d.
1/ 7/27	180/100	$CaCl_2$ 0 45 KI 0 6 t i d
2/14/27	166/112	
3/18/27	186/112	KI $CaCl_2$ āā 0 6 t i d
3/30/27	184/110	KBr $CaCl_2$ āā 0 6 t i d Nitroglycerin 0 0006 q i. d
4/13/27	196/112	Ca lact 4 t i d
4/20/27	192/100	Ibid.
5/ 4/27	198/130	Ibid
5/18/27	180/104	Ibid.
6/22/27	190	Ibid
8/10/27	130/70	Ibid
8/24/27	180/114	Ibid
9/ 7/27	160/110	Ibid.
9/28/27	150/100	Ibid
10/19/27	160/100	Ibid
11/ 2/27	176/106	Ibid.
11/16/27	214/122	Ibid

TABLE 4—*Continued*

Date	Blood pressure	Therapy
11/30/27	190/120	Ibid
12/21/27	150/100	Ibid
1/11/28	190/110	Bi subnitr 0 3 t i d
1/25/28	200/110	Ca lact 4 t i d
2/29/28	180/120	$CaCl_2$ 0 38, KBr 0 6 t i d
3/21/28	170/120	Ibid
4/18/28	164/100	KI, $CaCl_2$ 0 6 t i d
5/16/28	184/95	Bi subnitr 0 3, $CaCl_2$ 0 6 t i d
6/ 6/28	170/100	Ibid
6/27/28	172/110	Ibid
7/11/28	200/120	Ca lact 4 t i d

Blood

Date	Red cells	White cells	Hemoglobin	
10/22/26	4,920,000	9500	90	
3/31/27	5,640,000	4900	85	Guanidine 0 48

Urine

Date.	Albumin.	Sugar	Casts	Specific gravity	Phenolsulphonephthalein 15 min	30 min.	60 min.	Total
10/ 8/19	0	0	0	1016				
11/ 6/19	Trace	0	Hyal	1015				
11/13/19	0	0	0	1022				
12/ 8/19	0	0	0	1028				
10/27/21	0	0	0	1008				
12/28/22	0	0	Hyal	1015				
12/10/23	0	0	0	1014	20%	10%	8%	38%
3/ 6/24	0							
6/ 7/25	0	0	0					
2/18/27	++	0	Cell					
2/25/27	++	0	0	1021				
3/18/27	+	0	0	1015				

On October 18, 1921 fluoroscopic examination revealed a widened aorta and a heart shadow which extended to the left axillary line On October 27, 1921 it was noted that she complained of dimness of vision of left eye, of a feeling of heaviness all over the right side, especially in the extremities, of pain in the lumbar region of two months' duration which was aggravated by work and associated with palpitation, constipation unless laxatives were used, nocturia two or three times, and headaches In the spring of 1924 she complained of dimness of vision, vertigo, dyspnea, constipation, and had a herpes zoster involving the seventh and eighth right intercostal spaces

In November, 1925 the following complete history was taken and physical examination made

Present Complaint—Dull pain in the region of right axilla with radiation to shoulder, palpitation which is most marked when she lies down, dull continuous pain in region of left hip and lumbar region

Past Illnesses—Eight years before, in 1917, the patient awoke one morning with inability to see out of right eye, which was followed a year later by a "stroke," followed by diminished sensation and numbness on the right side, which had persisted For two years she had been conscious of a

choking sensation or stuffy feeling in the chest and rapid heart action on slight exertion For three years she had been constipated Some infected teeth were removed in 1922 At times there is some difficulty in holding urine and she gets up two or three times at night to urinate. She has had headaches and some vertigo almost constantly for years

In childhood she had croup mumps whooping cough measles tonsillitis typhoid fever and a broken right forearm As an adult she had pleurisy rheumatism in 1922 erysipelas in 1920 She had been pregnant seven times resulting in three miscarriages and four living children two of whom are now dead. Menopause occurred in 1920 but uterine bleeding appeared four times thereafter

Her father died of paralysis her mother of tuberculosis Two sisters living one of them not well two brothers dead one dying of measles

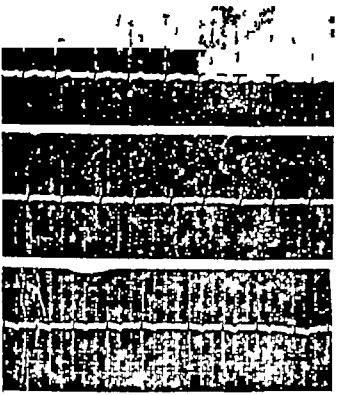

Fig 73 —1/6/26 Normal mechanism sinus arhythmia

Physical Examination —Head neck, and lungs were negative There was bulging over precordium right heart diameter 4 cm. to right and left heart diameter 12 cm to left of midline apex in sixth interspace. There were no murmurs but the first sound at the apex and the second sound in the aortic area were accentuated The rate and rhythm were normal The abdomen and extremities were normal. Both patellar reflexes were diminished

On January 6 1926 because of a suspected pulsus alternans an electrocardiogram was made (Fig 73) Her condition remained about the same until the spring of 1927 when she had more precordial discomfort and some symptoms of right heart failure A second electrocardiogram was made on

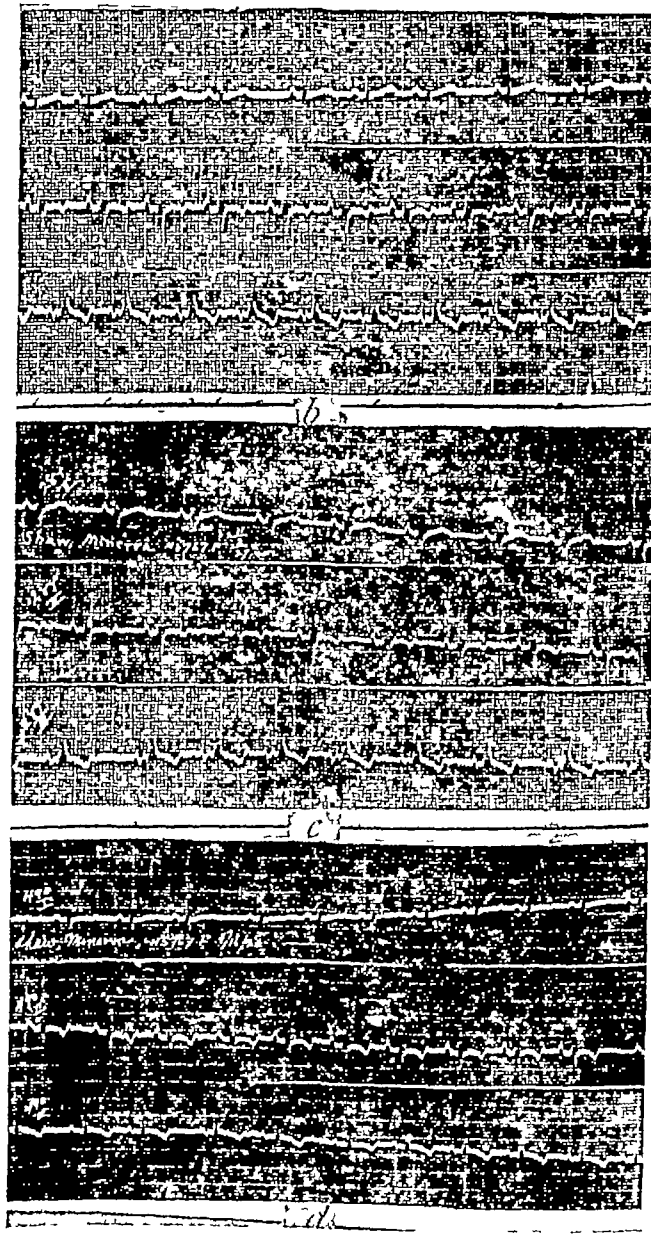

Fig 74.—*b*, 4/13/27 P2 diphasic and notched, T2, 3 inverted, QRS 0 1 sec , left heart preponderance
c, 11/2/27 P3, T2, 3 inverted, QRS 0 2 slurred
d, 6/16/28 P2 high, T2, 3 inverted, QRS 0 075, left heart preponderance

April 20 1927 (Fig 74 *b*) The cardiac pain was relieved by nitroglycerin The general condition has remained about the same except that vertigo has been more marked. Electrocardiograms were made November 11 1927 (Fig 74 *c*) and May 16 1928 (Fig 74 *d*) The clinical and *x* ray laboratory findings were as given in Table 4

This is a case of chronic hypertensive cardiovascular renal disease in which the vessels of the eye of the equilibratory apparatus and of the heart are especially involved and in which renal findings appear to be of relatively slight importance The fact that the eye condition has remained about the same over a period of years makes it appear that the condition there is stationary The electrocardiographic changes that took place between January 1926 and May 1928 are most illuminating and are illustrative of the value of the electrocardiogram in the careful study of this type of case In January 1926 there was marked sinus arhythmia slight slurring of the QRS in all leads and an inverted T in leads II and III In May 1927 the P R interval was 0 18 second P2 was very high and notched and P3 diphasic. The QRS interval was 0 10 second T was inverted in leads II and III and there was a marked left heart preponderance, good evidence of disturbed auricular conduction and of ventricular circulation involving more especially the right coronary branches

By November 1927 the P3 which in May was diphasic had become inverted indicating a progress of the pathologic condition In May 1928 P3 was again diphasic but otherwise the electrocardiogram is much the same so that it seems that the condition has not changed materially during the last few months. The high blood guanidin should be noted

Of course the prognosis is not good in this type of case. Calcium therapy seems to have kept the blood pressure at a lower average than did the types of treatment attempted before it was started. It has certainly caused the patient to feel better though this may be attributed in part to diet She is supposed to have been on a low salt low maintenance protein diet but it is doubtful if one in her station in life really is able for financial and other reasons to follow a diet with any degree of accuracy

It seems that this patient probably originally had an essential hypertension which as it became chronic caused sclerotic changes in the vascular system which as is so often the case in the chronic types of hypertensive vascular disease predominate in the vessels of the heart The relatively slight evidences of renal damage found in the presence of a hypertension certainly present for ten years in a case in which there is evidence of progressive changes in the cardiac vessels make it highly improbable that kidney pathology is of itself a primary etiologic factor in such cases

The progressive changes in the electrocardiogram of this and the preceding case (G F) are most interesting While both of them have had more or less cardiac pain neither has had the syndrome typical of coronary occlusion and yet the changes in the cardiogram are those that might be ascribed to such a lesion It is probable that there is a marked sclerosis of the coronary arteries in both and at necropsy there would probably be found areas suggestive of healed infarcts with almost completely or completely occluded arteries

In reviewing these 4 cases, it seems that there is no direct relationship between the height of the blood-pressure, systolic or diastolic, the degree of change in the retinal vessels, in the renal function, in cardiac size as shown by the 2 M plate, and the electrocardiogram Three of the cases have apparently been more or less improved both subjectively and objectively by the therapy they have had, and the fourth is better subjectively Results in other cases indicate that larger doses of calcium lactate, at least 8 gm , three times daily, are more effective in lowering pressure than the 4-gm doses used in these Calcium lactate in such doses has been found more effective than calcium chlorid in doses of 0 3 to 0 6 gm three times daily, alone or with ammonium or potassium chlorid, bromid, or iodid in average doses

In summarizing, the results, in these cases and the sixty or so more that have been under treatment in this clinic during the past five or six years, make it appear that a low salt, low protein diet, containing an abundance of carbohydrate, especially of greens, plus large doses of calcium salts, is beneficial in the treatment of hypertension In the majority of the cases there has been a very considerable fall in blood-pressure, with an increase when the calcium was stopped In practically all of them there has been marked subjective improvement This therapy appears to be of sufficient value to warrant its further use

CLINIC OF DR WALTER LINCOLN PALMER

ALBERT MERRITT BILLINGS HOSPITAL, UNIVERSITY OF CHICAGO

CERTAIN PHASES OF THE ULCER PROBLEM

THE cases to be presented here have been selected as representative of certain difficulties encountered in the diagnosis and treatment of gastric and duodenal ulcer

Case I.—The first is that of a man forty two years old who entered the hospital on the surgical service of Dr Phemister October 27 1927 complaining of epigastric distress of seventeen years duration

In 1910 when the distress first appeared it was localized to the epigastrium, appeared regularly one to two hours after eating was absent in the morning before breakfast and was regularly relieved by food taking or by soda. The diagnosis of duodenal ulcer was made and medical treatment consisting of dietary restriction and occasional alkaline powders instituted The distress disappeared temporarily but recurred within a few months With each recurrence of the distress usually in the Spring and the Fall the diet was resumed and relief obtained There is a history of his having passed black tarry stools four or five times during such periods but no other evidence of massive hemorrhage. In 1926 a penetrating lesion of the duodenum was reported on x ray examination and some months later this was said to have shown marked improvement In this way the patient got along fairly well until April, 1927 when he developed a different type of abdominal distress a cramp-like pain in the peri-umbilical region coming on at irregular intervals but usually one to three hours after eating not relieved by a teaspoonful of baking soda, but usually relieved by food taking This pain occasionally radiated through to the right scapula but was not accompanied by vomiting and was never severe enough to require a hypodermic. The attacks were occasionally followed by clay-colored stools jaundice, and very dark urine The remainder of the history was relatively unimportant

Physical examination revealed no abnormality other than some tenderness in the right lower quadrant, and marked tenderness and rigidity in the right upper quadrant The temperature and pulse were normal and the blood pressure was 140/60

The laboratory work showed a normal blood picture a negative blood

Wassermann, normal urinalysis, normal gastric analysis with a maximum free acidity in the fractional alcohol test-meal of 64, normal stools which on a meat-free diet were negative for occult blood by the benzidin test

x-Ray examination of the stomach had been made in three other laboratories and had revealed a definite duodenal deformity interpreted as that of duodenal ulcer Two of the laboratories had also diagnosed a non functioning gall-bladder containing stones

The patient was operated on by Dr Phemister on October 25, 1927, under the preoperative diagnosis of cholelithiasis and probable duodenal ulcer The gall-bladder was found to be adherent to the second portion of the duodenum with adhesions involving the first portion also, the wall of the gall bladder was thickened, edematous, and contained much fibrous connective tissue Numerous stones of various size were present The duodenum and stomach were explored, but no evidence of ulcer found except the adhesions, and these appeared to be the result of the adjacent cholecystitis Gastro enterostomy was considered, but because of the possibility that all of the symptoms could be explained on the basis of the cholelithiasis and chronic cholecystitis, the idea was abandoned The gall-bladder was removed and the abdomen closed with drainage Recovery from the operation was prompt and satisfactory

The patient was discharged from the hospital with instructions to eat a full and general diet This he did, and overdid, in an effort to compensate for the years of dietary restriction In July, 1928 he returned to the clinic complaining of distress about the navel and extending across the abdomen, coming on at irregular intervals and apparently relieved by a bowel move ment Stool disturbance had been marked, the consistency varying from mushy to formed, but of small caliber Physical examination revealed tenderness along the colon The distress was thought to be that of a bowel disturb ance due to dietary overindulgence and indiscretion A soft diet was pre scribed together with tincture of belladonna and powders of calcium car bonate, calcium phosphate, and bismuth subcarbonate

Marked improvement followed, but when the patient returned six weeks later it was found by Dr Ortmayer that his distress occurred regularly at 4 and 9 P M , was quite severe for an hour or an hour and a half, and was re lieved in half an hour or so by a bowel movement, by a powder, or by rest and heat to the abdomen The effect of food taking and of baking soda had not been tried An "acid test" on September 8, 1928 gave the following result

10 10	A M	Stomach empty No distress present, 200 c c. 0 5 per cent HCl injected into the stomach through a Rehfuss tube
10 20	"	No distress
10 27	"	Dull pain appears in midepigastrium similar to regular complaint
10 30	"	Dull pain persists
10 40	"	Additional 200 c c 0 5 per cent HCl injected
10 45	"	Pain decreasing in severity
10 55	"	Pain more severe
11 00	"	Pain still increasing in severity
11 10	"	Test discontinued because of extreme typical pain in epigastrium

11 20 A M Stomach emptied 300 c.c of pale green liquid being obtained with a free acidity of 94 total 97 The relief from the distress was almost complete within ten minutes
11 30 Sodium bicarbonate 8 gm (5ii) given by mouth
11 35 Relief complete

Fluoroscopic examination of the stomach the same day revealed a definite deformity of both curvatures of the duodenal bulb A motor meal showed moderate retention after seven hours. The stools were negative for blood by the benzidin test.

The diagnosis of active duodenal ulcer seemed inescapable and after consultation with Dr Phemister medical management was agreed upon The patient remained in the hospital for three weeks during which time he was given antacid ulcer therapy of the type first recommended by the late Dr B W Sippy On the usual powders it was found that his gastric free acidity was not neutralized during the day or in the evening and hence the calcium powders were increased to calcium carbonate 1 2 gm (gr xx) sodium bicarbonate 2 gm (gr xxx) and the magnesia powders to heavy magnesium oxid 0 6 gm. (gr x) calcium carbonate 0 6 gm. (gr x) and sodium bicarbonate 1 2 gm. (gr xx) On this management with hourly powders and with the hourly milk and cream régime the gastric free acidity was regularly found to be neutralized during the day and in the evening The distress disappeared immediately on entering the hospital and there has been no recurrence to date

On October 29 1928 the acid test was repeated with the following result

9 40 A M No distress Stomach emptied 140 c.c. yellow liquid free HCl 0 total HCl 9
9 45 200 c.c. 0 5 per cent. HCl given per Rehfuss tube.
10 15 200 c c. 0 5 per cent HCl given per Rehfuss tube.
10 45 200 c.c. 0.5 per cent HCl given per Rehfuss tube
11 15 No distress whatsoever at any time during the test. Stomach emptied 80 c c. liquid free HCl 78

Fluoroscopic examination of the stomach on this date again enabled one to visualize a constant duodenal deformity such as might be produced either by ulcer or by adhesions but no crater was demonstrable

A motor meal of coarse food was eaten November 23 1928 On direct aspiration seven hours later no gastric content was obtained and the washings returned clear

The patient has continued his treatment faithfully since leaving the hospital has had no recurrence of his distress, and has aspirated his own stomach at 9.30 P M sufficiently often to know that under this régime it is practically empty at bedtime, and no free acidity is present

Discussion.—The combination of cholelithiasis and chronic duodenal ulcer is not a very unusual one, and this case is not presented as an illustration of that point, but for other reasons The response of the ulcer to medical management was typical,

but the whole question of therapy will be reserved for discussion later

One very important question is brought forward, however, by this case, namely, that of periduodenitis secondary to cholelithiasis Duval, Roux, and Beclere of the French school have written on this subject in recent years and reported cases similar to this in which there were recurrences of distress of the so-called "pyloroduodenal syndrome" type after cholecystectomy In some of these cases the duodenum appeared normal at the time of operation, but months or years later distress reappeared Upon radiologic examination deformities of the duodenal bulb were found which were interpreted as those of periduodenitis rather than of duodenal ulcer Subsequent laparotomy confirmed the finding of peribulbar adhesions without giving other evidence of duodenal ulcer Gastro-enterostomy with pyloric occlusion was followed by relief from the distress Upon such evidence these writers rejected the diagnosis of ulcer and accepted as proved the hypothesis that the distress was due to periduodénitis per se This conclusion seems to us to be unjustified

It is generally recognized that chronic gall-bladder disease may involve the duodenum by contiguity in an inflammatory or cicatricial mass, and that all grades of duodenal stenosis may result therefrom It should be remembered, however, that such stenoses, essentially extraduodenal, are not characterized by pain but by nausea and vomiting This characteristic picture due to pyloric obstruction alone, or to obstruction in the first few inches of the duodenum, is not infrequently seen in healed stenotic ulcers, in malignant disease of the pylorus, and occasionally, although much more rarely, as the result of adjacent gall-bladder disease or adhesive tuberculous peritonitis Such being the case it is difficult to see why the similar process of periduodenitis should be painful, for in the type under discussion the mucosa is not involved and the lesion is practically extraduodenal

It is a well-known fact that the presence of ulcer cannot be excluded by palpation of the stomach and duodenum during a laparotomy This is proved most conclusively by the finding of

gross ulcers in resected specimens which had been entirely overlooked at the time of operation The task is especially difficult when the bulb is surrounded by and involved in adhesions. In fact, such adhesions are in themselves very suggestive of ulcer

Recognizing, then, the impossibility of excluding ulcer by palpation at the time of operation, and the impossibility also of stating accurately from radiologic evidence as to whether a duodenal deformity is due to an intraduodenal or an extraduodenal lesion (assuming the absence of an "en face" niche and direct visualization of the crater) we must look for more direct evidence of ulcer in this case which is entirely similar to the reported cases of distress attributable to periduodenitis

The first suggestion of this is to be found in the history The original epigastric distress with which the patient had been troubled bore the earmarks of ulcer It was absent in the morning before breakfast, appeared one to two hours after food-taking, and was regularly relieved either by food-taking or by soda It then came to have also the classical properties of chronicity and periodicity Such a typical picture as this speaks very strongly for ulcer regardless of the presence of complicating conditions such as chronic gall bladder disease

A second point strongly suggestive of ulcer is the prompt and so far permanent relief on antacid ulcer therapy It is difficult to conceive of an extraduodenal or an extragastric lesion in which the pain would be so quickly and completely relieved by this form of treatment

The third piece of evidence for ulcer is to be found in the "acid test" Before the institution of ulcer therapy the "acid test" reproduced the patient's typical distress in a severe form Seven weeks later it produced no distress whatsoever The reasonable inference is that something has happened in the interim to alter conditions Presumably the alteration has taken place in the mucosa, and not in the serosa or periduodenal tissues, for we know of no medical means of influencing them directly The only known forms of change in the duodenal mucosa associated with such types of distress are ulcer and the diffuse inflammatory process, duodenitis, which is usually limited to the

mucosa The relationship between ulcer and duodenitis cannot be discussed here, but in this case the chronicity, the extensive periduodenal adhesions, and the definite deformity of the bulb as seen roentgenologically favor the diagnosis of duodenal ulcer rather than that of duodenitis

Perhaps in time it will be shown that periduodenitis per se, without any alteration of the mucosa, may produce other symptoms than the nausea and vomiting of stenosis, but at the present time there is very little, if any, proof thereof It is our belief that most of the reported cases of distress attributed to stenosing periduodenitis are in reality cases of duodenal ulcer

Case II —The second case is that of a male thirty-seven years of age who entered the Billings Hospital first on August 8, 1928, complaining of severe abdominal distress located chiefly in the epigastrium with some radiation toward the lower abdomen This distress had the typical earmarks of an ulcer distress in that it came on two or three hours after eating and was regularly relieved by food-taking or by soda It had been present for about three years, during which time various dietary régimes were tried but without bringing about any definite remission in the condition There is no other noteworthy information in the history The physical examination was essentially negative The blood Wassermann and Kahn tests were normal, the blood-picture was normal, urinalysis normal, stool analyses were negative for occult blood by the benzidin test, the Ewald test-meal showed a free acidity of 42 and a total of 64, the motor meal showed no gastric retention at the end of seven hours, the serum bilirubin was 0 865 unit, the basal metabolic rate was 1 5 per cent x-Ray revealed no evidence of foci of infection about the teeth or of urinary calculi, a normally functioning gall bladder using the intravenous Graham-Cole method, and a constant deformity of the duodenal bulb with very little narrowing of the lumen but with persistent fleck formation which was interpreted as visualization of the crater of a duodenal ulcer The acid test was as follows

2 50 P M Stomach aspirated Forty c.c. liquid obtained Free acidity 31 Total acidity 46 No distress present, 200 c c 0 5 per cent HCl injected into stomach

2 57 " Slight gnawing epigastric pain appears

3 03 " Pain increasing, cramp-like in character

3 35 " Gnawing epigastric pain identical in type and location with patient's typical distress continues, 200 c c 0 5 per cent HCl injected into stomach

4 12 " Typical distress continues, 200 c.c 0 5 per cent HCl injected into stomach

4 55 " Typical distress continues Stomach emptied, 350 c c. liquid Free acidity 93 Total acidity 99

5 12 " Very slight gnawing pain still present

The diagnosis of uncomplicated duodenal ulcer was made and the patient placed at once on Sippy management The distress disappeared immediately but frequent aspirations showed that the free acidity was not neutralized by the usual powders consisting of calcium carbonate 0 6 gm (gr x) and sodium bicarbonate 2 gm. (gr xxx) alternating with heavy magnesium oxid and sodium bicarbonate each 0 6 gm. (gr x) In order to accomplish complete neutralization of the free acidity it was necessary to increase the powders to the following Calcium carbonate and sodium bicarbonate each 2 gm. (gr xxx) and heavy magnesium oxid 0 6 gm. (gr x) calcium carbonate 0 9 gm (gr x) and sodium bicarbonate 2 gm (gr xxx)

After three weeks of treatment the stomach was again x rayed and the constant fleck formation in the duodenal bulb found to be still present The acid test was repeated with the following results

4 15	P M	Patient has no pain Stomach aspirated
4 20		200 c.c. 0 5 per cent HCl injected into stomach
5 06		No pain 200 c.c. 0 5 per cent. HCl injected into stomach.
5 10		Slight gnawing pain in region of navel
5.21	'	Distress ceases.
5 29	"	Pain reappears.
5.36	"	Distress ceases
5.39	'	200 c.c 0 5 per cent HCl injected into stomach
5 49		Gnawing pain in region of navel reappears
5 55		Pain ceases
6 10	'	No pain 400 c c. liquid aspirated from stomach

The patient was then discharged from the hospital on the three meal a day schedule with hourly milk and cream feedings and the same hourly powders as those used in the hospital Six weeks later the stomach was again x rayed the duodenal deformity again seen but the en face ' niche or fleck formation could not be demonstrated The acid test was repeated with the following results

9 10	A M.	No distress Stomach emptied 45 c.c. liquid Free acidity 60 Total acidity 63
9.25	'	200 c.c. 0 5 per cent. HCl injected into stomach
9 45		No distress 200 c.c. 0 5 per cent. HCl injected into stomach
10 15		No distress 200 c.c. 0 5 per cent. HCl injected into stomach
10 45		No distress at all. Stomach emptied 360 liquid. Free acidity 108 Total acidity 110

The patient is now back in his regular occupation as a mail clerk, working very hard and at irregular hours but nevertheless continuing his management He has gained 9 pounds in weight and there has been absolutely no recurrence of his distress

Discussion—At the time of admission the patient was having a regularly recurring and very severe epigastric distress His behavior was that of a man in agony, but it was in many respects suggestive of a psychoneurosis The institution of ulcer manage-

ment was followed by immediate relief from the abdominal distress and all trace of the psychoneurosis disappeared This point deserves emphasis, for one so commonly sees patients passed from physician to physician, and from clinic to clinic, labeled as "neuros" until finally some underlying condition, such as an atypical duodenal ulcer, is found and properly treated The psychoneurosis then disappears in whole or in part

The "acid tests" illustrate the various results which may be obtained In active periods of distress the typical response is that shown in the first test If, however, the spontaneous distress is mild, the response may be similar to that in the second On the other hand, if there has been no spontaneous distress for a day or two, the "acid test" is likely to be negative, as in the third result When positive the test is often useful as a diagnostic procedure, but when it is negative it is of much less significance It is also important and characteristic, that in the second test after three weeks of management the distress produced by the acid was of much less severity than in the original one Furthermore, it did not appear until after the second injection In other words, a decrease in the sensitiveness of the pain-producing mechanism was indicated by a lessening in the severity of the pain, and a prolongation of the latent period In the third test the ulcer was either totally insensitive or the latent period exceeded the duration of the test This is an absolutely characteristic response to antacid ulcer therapy The length of time required in uncomplicated ulcer for the acid test to become negative varies greatly, but in my experience it eventually does so in all cases

It is usually not possible to correlate the healing of a duodenal ulcer with an alteration in the deformity as seen roentgenologically except in the cases in which the crater is visualized Unfortunately, this does not occur as frequently as one might wish Here, however, the crater was actually visualized at the time of the first two acid tests and then six weeks later when the acid test was negative, the crater was not demonstrable These two points together with the complete relief of symptoms certainly constitute strong evidence of healing of the ulcer Berg

has noted that the symptoms of ulcer disappear before the niche disappears roentgenologically and it has been my observation that the acid test becomes negative at a time when the crater is still demonstrable radiologically In other words, a negative acid test usually betokens healing of an ulcer but it does not imply that the process is complete Similarly, the disappearance of spontaneous pain may indicate merely that the healing proc esses are predominating The same statement applies to the disappearance of a radiologically demonstrable crater, for surgical experience has shown that this may occur before healing is complete

These facts are of great importance from the standpoint of the duration of treatment and bring us to a consideration of that question Obviously treatment should be continued until the ulcer is well healed, but at the present time there is no means of knowing definitely whether or not the process is com plete. The best evidence available—the relief from symptoms, the negative acid test, and the disappearance of the crater as seen roentgenologically—is unreliable Clinical experience has shown that as a rule it is not safe to discontinue ulcer therapy in less than a year and often a longer period is necessary Occa sionally one sees at autopsy of patients dying from other causes ulcers which have healed completely within a few weeks, but the usual chronic ulcer requires several months for the process

Case III.—The third case is that of a male thirty five years old who entered the Out patient Department on March 10 1928 complaining of abdominal distress of eight years duration Originally the distress had centered in the epigastrium, radiating through to the back, had appeared about one hour after meals and had regularly been relieved by food taking Soda had also given some relief but apparently of brief duration He spent two months in a hospital in England at this time and was treated with a bland diet and powders The distress disappeared and did not reappear until two years later at which time April 1922 a laparotomy was performed. An appendectomy and a gastro-enterostomy are said to have been done but no record of the findings is obtainable This was followed by a two-year period of freedom from distress but in 1924 pain reappeared of a somewhat different type than before located lower at about the level of the navel and radiating to the lower abdomen very severe at times and frequently accompanied by nausea and vomiting Hunger was present all the time but the patient was afraid to eat The pain was not relieved as readily by food taking as the

original distress had been, soda was much less effective, and vomiting also gave only partial and incomplete relief In fact, the patient found that more relief could be obtained by lying flat on his back than in any other way Nausea and vomiting frequently occurred independent of pain and for periods of two or three weeks at a time would constitute the major complaint This state of affairs lasted until the patient's admission to the hospital, not materially affected by two or three ambulatory forms of therapy tried in the interim In other respects the history was unimportant

Physical examination was essentially negative except for extreme tenderness in the region of the navel The blood and spinal fluid Wassermann and Kahn tests were negative, the spinal fluid cell count was 15 cells per cubic millimeter, the Ross-Jones test for globulin was faintly positive, and the colloidal gold curve 0001110000, urinalysis was normal, the blood-picture normal, stool analysis was negative for occult blood by the benzidin test, the Ewald test-meal returned 90 c c of gruel with a free acidity of 50 and a total of 57, a fractional alcohol test-meal returned in each aspiration only a few cubic centimeters of bile-colored liquid containing no free acid *x*-Ray revealed no evidence of urinary calculi, a normally functioning gall-bladder with no evidence of stones, using the Graham-Cole technic, a large duodenal bulb with no evidence of deformity either in the bulb or in the stomach, no evidence of a gastro-enterostomy opening, but the third portion of the duodenum was very large and much dilated as though some obstruction existed beyond it

The history was typical of a gastrojejunal ulcer, the recurring distress being more severe than the original, located lower in the abdomen, and less definitely related to or relieved by food-taking The attacks of nausea and vomiting unassociated with pain were very suggestive of the so-called "vicious circle," perhaps due to obstruction of the distal loop of jejunum *x*-Ray had failed to reveal evidence either of gastro-enterostomy or of gastrojejunal ulcer, but the fractional alcohol test-meal had typically returned only a few cubic centimeters of liquid obviously containing a large amount of yellow bile The diagnosis of gastrojejunal ulcer with a non-functioning gastro enterostomy and a so-called "vicious circle" was made The patient contracted an upper respiratory infection and hence did not enter the hospital for operation until nearly six weeks later, during which time he remained on a soft diet and was entirely free from pain, although not from nausea

Laparotomy was performed April 21, 1928 by Dr L E Dragstedt, who made the following report of his findings "The scar of the duodenal ulcer was seen in the anterior wall of the duodenum about 1 inch from the pylorus The gall-bladder was explored and found normal A posterior no-loop gastro-enterostomy was found and the jejunum was sharply kinked at the gastro-enterostomy opening The line of anastomosis was cut through and the stomach separated from the jejunum The stomach was then closed A thick fibrous band was attached to the jejunum at the line of anastomosis and beneath this apparently a healed jejunal ulcer The hole in the jejunum was closed transversely" The patient recovered quickly from the operation and was discharged from the hospital May 10th with instructions to eat a normal diet The old duodenal ulcer had appeared healed at laparotomy and the

gastro-enterostomy had been undone Hence no indication for dietary restrictions could be seen

On July 4th the patient developed what appeared to be neuritic or arthritic pain in the back between the shoulders and extending down into the lumbar region A mustard plaster was applied with considerable relief He reported in the Out patient Clinic July 9th and was told to continue the therapy which he himself had instituted At the time of his next visit two weeks later July 25th his distress had grown more severe and was accompanied by epigastric pain No definite relationship to food taking had been observed and the effect of food taking and of soda upon the distress had not been tried *x* Ray examination of the thoracic spine did not reveal evidence of a lesion An acid test July 27th reproduced the patient s typical distress in the back and also some in the epigastrium. Fluoroscopy of the stomach the same day showed a constant fleck in the duodenal bulb which remained in the same location and which could not be wiped away when barium was pressed from the cap in other words, it had the appearance of barium lodged in the crater of an ulcer

The patient was then hospitalized for observation During the first few days on an unrestricted diet the distress was found to possess all of the characteristics of an ulcer pain as regards relationship to meals and relief from food taking and alkalis. It was unusually severe in the back, and at times was present only in the lower thoracic and upper lumbar regions posteriorly with no radiation or distress anteriorly An acid test July 31st produced severe pain in these areas but slight and transitory epigastric pain Ulcer management was begun August 1st with prompt and complete relief from the distress. The following powders were required to neutralize the high-grade acid secretion Calcium carbonate 2 gm. (gr xxx) sodium bicarbonate 3 gm. (gr xlv) alternating with heavy magnesium oxid 0 6 gm. (gr x) calcium carbonate 1.2 gm. (gr xx) and sodium bicarbonate 2 gm. (gr xxx) On August 17th the acid test failed to produce any distress, except nausea in one and a half hours Fluoroscopy revealed a constant deformity of both curvatures of the bulb and a constant but faint fleck in the same position as that seen July 27th The patient was discharged August 22d with the usual instructions.

He returned to his work as guard of a payroll car with irregular and long hours and did not follow out the instructions given at all. In fact he modified his management to the extent that it came to consist of three or four meals daily at irregular hours five or six powders daily and no milk and cream. The distress recurred but was always relieved by a powder The situation was then talked over frankly with the patient and he was led to see that it would be possible for him to follow his treatment accurately while at work. This he is now doing and is reporting regularly with a record of the amount and reaction of his nightly aspirations. The distress has disappeared again and he is getting along very well

Discussion —This case has many interesting phases, but only a few of them can be discussed at this time The distress attrib-

utable to the gastrojejunal ulcer was typical of that lesion, as has been stated The nausea and vomiting not accompanied by pain are to be looked upon as motor disturbances associated with the gastro-enterostomy which might not have occurred if an entero-enterostomy had also been done The pain in the back which recurred was undoubtedly ulcer distress, atypical in location The usual epigastric type of ulcer distress frequently radiates through to the back just as does the pain of gall-bladder colic and it may be felt more severely there than in the epigastrium Occasionally the pain is noticed only in the back Such atypical forms of ulcer pain are not at all rare and may occur either with a gastric or a duodenal lesion Formerly this back pain in ulcer was thought to occur only in cases in which the ulcer had extended into the pancreas but this is not the case for it has been noted in instances in which at operation the lesion was found to be limited to the wall of the stomach or duodenum

The reactivation of an apparently healed duodenal ulcer has not infrequently been observed as a sequel to the closure of a gastro-enterostomy opening It illustrates in the first place the efficacy of gastro-enterostomy in promoting the healing of a duodenal ulcer It has also a very important bearing on the more fundamental aspects of the ulcer problem, for the healing as a result of a simple gastro-enterostomy and reactivation after closure of the gastro-enterostomy stoma strongly favor the view that the gastric chyme is an important factor both in the pathogenesis and chronicity of ulcer These problems have been subjected to much experimentation and more discussion but are still unsolved

The most important and immediate question in this case is that of the therapy It was known at the time of the laparotomy that to undo the anastomosis was to court reactivation of the original lesion, but the gastro-enterostomy had caused more trouble than the primary ulcer To do another gastro-enterostomy meant the likelihood of another gastrojejunal ulcer as in the case recently reported by Thompson and Steward in the *British Journal of Surgery* in which four gastro-enterostomies were done one after the other over a period of

sixteen years with the old ulcer healing each time and a new one forming The chance of reactivation of the duodenal ulcer did not appear to be greater than that of another gastrojejunal ulcer if another gastro-enterostomy were done Furthermore, it was felt that should the old ulcer recur in the non stenosed duodenum, it could be handled much more easily medically than could a gastrojejunal ulcer It was the more conservative procedure and one must remember that there is no known form of medical or surgical treatment of ulcer short of total gastrectomy which affords an absolute guarantee against recurrence of the lesion

There are many who would say at once that a subtotal gastrectomy should have been done at the time of the last operation, or that it should be done now We are not in accord with this idea and would not be even if the operation itself could be performed without any mortality rate, for the danger of morbidity which it carries does not justify it Balfour has recently reported a series of fifty eight recurrences following subtotal gastric resection It had been my intention to present another here, but time does not permit. In brief, ulcers do recur following gastric resection, and recur again following further resection Such recurrences seem to be much more difficult to treat medically than the original ulcer, and surgically there is nothing to do except to resect more and more of the stomach with the hope of finally effecting a cure, although this may not occur until total gastrectomy has been accomplished Usually the patient decides to bear the ills he has, and goes on through life more of an invalid than he was originally It must be admitted that in the majority of cases a complete and permanent cure is obtained, but unfortunately the minority, those with recurrence, do constitute a definite percentage These individuals are in a difficult and sorry plight indeed, and hence partial gastrectomy should be recommended only as a last resort

CLINIC OF DR JESSE R GERSTLEY

NORTHWESTERN UNIVERSITY MEDICAL SCHOOL AND
MICHAEL REESE HOSPITAL

CHOREA

THIS little girl is ten years old

One year ago a rat ran across the floor of her apartment and touched her foot Immediately afterward she became very nervous A local physician diagnosed chorea, and sent her to the hospital There she stayed for about six weeks and was discharged as cured For one year she remained well, but now the mother brings her back with a recurrence of the symptoms A rat running around an ash can in the alley seemed to precipitate this attack Our young lady seems to have a rat psychosis

The rest of the history is of no great clinical importance Tonsils and adenoids were removed two years ago Some teeth were extracted last year There is some nervous instability in the relatives on the mother's side

Examination —As you notice, this little girl is somewhat overweight We shall return to this later As she lies on the examining table, notice the jerky movements of the extremities, especially of the right side. For some unknown reason almost all choreas are worse on one side than the other The child herself will tell you on which side the disease is severest. Children usually describe the sensation as a feeling of weakness or nervousness in one or more extremities When we examine these jerky movements more carefully we can divide them into two types

1 Lightning like contractions of isolated muscle groups, groups which we cannot cause to contract voluntarily

2 Incoördinate, awkward movements of the larger muscle groups Notice the grotesque gestures when I ask this little girl

to shake hands with me—or to convey this cup of water to her mouth A combination of the two types of movement is seen when we ask her to open her mouth and put out her tongue The mouth is opened with a very definite grimace The protruded tongue is tremulous and full of minute quivering muscular contractions

A number of other signs may be of interest to you In addition to the general increase in all reflexes there is a special phenomenon shown by the knee-jerk A single tap causes a decided response If now we tap the affected extremity (in this case the right) repeatedly, the leg is thrown into a sort of tetanus and held in extension for a number of seconds Another interesting clinical observation is the following When I ask this child to squeeze my hand with her right hand, you notice there is little associated movement in her left hand When we reverse the test and she squeezes my hand with her left, there are decided associated movements in her right In other words, these associated movements appear in the side most involved The Czerny sign may be present When a child takes a deep breath the abdominal wall, due to the increased abdominal pressure from diaphragmatic contraction, is usually protruded In chorea the wall often is drawn in I do not regard this as a sign of major importance

The most valuable aid in diagnosing questionable cases I have found to be the following Try the effect of mental excitement upon the muscular movements If afflicted with only a mild case, the patient lying quietly in bed may show no symptoms whatsoever, but under nerve strain he will develop typical choreiform muscular movements In the case of this little girl the condition is severe enough to preclude absolute muscular quiet, but still note the effect of mental activity I shall give our little friend some problems in arithmetic just a bit difficult for her The muscular response under this mental stimulus is most significant Whenever I am in doubt as to diagnosis I have found this the most valuable of all tests

Etiology and Treatment—You may be startled at my grouping these two great subjects under one heading However, the

reason is simple We do not know the actual etiology of chorea There are a number of hypotheses In a like manner there are many different treatments By trying various treatments it might be possible to get some clue as to the etiology

The *prevailing* hypothesis is to link the etiology of chorea with that of rheumatism The occurrence of chorea in close association with tonsillitis, endocarditis, and rheumatism has led many observers to be quite positive in their statements that chorea is a rheumatic disease

The *prevailing* treatment of chorea is, first and foremost, rest in bed Secondly, come a variety of drugs If chorea is rheumatic the salicylates are logical If the child is being exhausted by muscular activity, luminal or bromid is indicated I am not very fond of luminal, having seen a number of cases of poisoning following its use At times I have had the impression that after luminal is stopped the symptoms become worse than if no medicine had been given Bromides are always of value As regards any specific drug treatment, Fowler's solution in increasing doses always was the standard remedy About fifteen years ago many pediatricians came to the conclusion that Fowler's solution was valueless From that time on in our own wards we have followed the practice of keeping the children in bed for five or six weeks, using medication only when indicated The essential, of course, is to keep them as isolated from the other children as possible and to come as close to mental and physical rest as is feasible in the ordinary children's ward During these years the neurologists have maintained that arsenic is of value and that it should be used in larger doses than those employed by the pediatricians, namely, injections of sodium cacodylate

The prevailing theory that chorea is related to the rheumatic infections has led to the careful search for foci of infection In addition to the above treatment, the modern pediatrician searches for the usual diseased tonsils and asks special advice as to the treatment of infected sinuses and teeth This is done as a routine

I first became more than ordinarily interested in the study of chorea some years ago when Dr E C Rosenow announced that he had isolated a streptococcus which caused typical chorea in

rabbits, could be isolated from the brain of rabbits, and could be passed through series of rabbits, always producing choreiform symptoms He prepared a serum against this organism and was kind enough to permit me to try it I made quite a careful study at that time [1] In connection with our resident physician I tried it on a number of cases and did considerable work along the lines of agglutination We found the serum to have no clinical value whatsoever It had no effect on the course, the complications, or the recurrences of the disease This, of course, does not speak for or against the theory of the streptococcus origin of chorea Let me digress one moment to explain this more thoroughly

During an epidemic of meningitis at Coblentz I was in charge of much of the contagious work for the army of occupation The soldiers were treated in the usual way with lumbar punctures and injections of antimeningococcus serum In spite of the most patient devotion, the sleepless nights of the attendants, the appeal of humanity that gave to duty an energy superhuman, our treatment was of no avail I cannot describe to you the feeling of utter helplessness, of absolute despair when day after day I saw these boys getting progressively worse independent of anything and everything that could be done Utterly uninfluenced by any form of treatment they died one by one In desperation I began to wonder if the serum itself was of no value I had heard that somewhere in France the Pasteur serum was giving good results, and so, after much argument and a final appeal to the higher authorities, we were able to procure some

The new serum worked like magic From being up night and day with the hopelessly sick and dying we were able to effect cures with a few intraspinal injections One hundred per cent fatality changed to 100 per cent recovery From a frightful pestilence the disease changed over night to a relatively mild infection A few doses of serum and the ailment terminated almost by crisis So startling were these results that they warranted further investigation I was fortunate in arousing the interest of Dr Robison, chief of the laboratories, and we decided to check

[1] Gerstley, J R, and Wilhelmj L J Amer Jour Dis Child, 33, 602, 1927

up the agglutinating properties of the various sera We found that the American serum agglutinated a number of different strains of meningococci But it *did not* agglutinate the *one* strain of organism that was present in the spinal fluid of the American boys in Coblentz The Pasteur serum agglutinated all strains Hence, the difference between 100 per cent mortality and 100 per cent recovery in this particular epidemic was due to the fact that that *one particular strain* of meningococcus was not included in the preparation of the serum

This experience with meningitis[1] was one of the great experiences of my medical life and one which I will never forget Hence if a serum does not give results in the treatment of a disease it is necessary to search still deeper before coming to final conclusions

In the case of chorea serum, then, we attempted to determine whether there were any specific organisms in the tonsils, nasopharynx, or abscessed teeth of these chorea children and whether the serum agglutinated any particular one of these organisms Our results were very unsatisfactory We found no specific type of streptococcus uniformly present, nor did we find the serum showed any specific agglutinating properties Hence we must conclude that the argument in favor of a definite streptococcus chorea is still unproved

In the course of this study, to determine whether the serum had any effect upon recurrences or complications, I organized a chorea clinic Such a grouping of cases gave an opportunity for a number of other investigations First, knowing that there is some sort of relationship between undernutrition and general nervousness, I tried the effect of increasing the weight of these youngsters But as our little patient here so ably demonstrates, weight is not a deciding factor Chorea occurs in the overweight and underweight alike In some of the under nourished children an increase in weight occurs simultaneously with an improvement of the disease, but this increase is the *result* of improvement and not the *cause*

The text books state, and I have found it true in my cases, that

[1] Robison J and Gerstley J R Jour Amer Med Assoc. October 1919

there seems to be some increase of the disease in spring Could chorea in any way be related to the tetany of infants which is due to a blood calcium deficiency developing after the long months of insufficient sunshine? We tried the electrical reactions and found that in many cases there is an increase of nervous irritability But, lo and behold, when we came to make blood calcium and phosphorus determinations, we found them to be normal In a like manner we got no striking improvement following the use of the ultraviolet ray So, unquestionably, chorea cannot be due to a calcium deficiency in the blood

The fact that these patients show such increased emotional reactions would lead one to think of the thyroid We made a number of metabolism tests, but the results were within the realm of the normal, the highest being plus 10 to plus 12 After a period of rest the rate dropped a little, but certainly there is nothing in these determinations to incriminate the thyroid

It is fashionable nowadays to talk of the glands of internal secretion A few men have given injections of adrenalin and reported good results, particularly in chronic cases We have seen nothing striking from adrenalin therapy When we were working with the calcium hypothesis, we treated some children with parathyroid extract (parathormone of Lilly) This also was disappointing

We have tried the effect of behavior[1] study on a number of children It occurred to us that the symptoms were made worse by uncongenial home environment, by irascible parents, and by bullying older brothers and sisters In such cases placing the child in a more congenial atmosphere proved of benefit But, of course, this is symptomatic treatment, and I would not go so far as to say that chorea was primarily caused by the nervous tension of the household, although at times I have been suspicious that this might be the case

At present we are pushing our studies along different lines No one has had the temerity to suggest dietetic influences in chorea The studies of the ketogenic diet in epilepsy might possi-

[1] Gerstley, J R Chorea A Brief Clinical Study with a Suggestion for Further Treatment, Ill Med Jour , 1928

bly have some application to chorea, not in any way as influencing the causative factor, but purely as a symptomatic treatment The resulting increase in the ketone bodies might act as a sedative just the same as bromid or luminal This is just a hypothesis but we are trying it out on a number of patients Our results will be reported in detail later

Another line of activity is the new serum recently introduced by Small Small, just as Rosenow, thinks he has found the organism of rheumatism and has prepared a serum against it He was kind enough to let us have some and we used it in a number of cases Up to the present the results are not very satisfactory On the other hand, some men think they have met with success especially in a few cases of rheumatism In our few cases the chorea was absolutely uninfluenced However, we have not studied a number sufficient to submit a convincing report

The Relation of Chorea to Rheumatism.—The cut and-dried opinion is that chorea belongs to the rheumatic syndrome To dispute such a theory takes considerable courage I am not prepared to dispute the hypothesis, but I must confess that I am beginning to have some doubts upon the subject In the first place, there is always a history of nervous instability in the family of chorea children and many times careful questioning will show that the mother herself suffered with chorea when a girl Thus, there is a definite hereditary element Again, the campaign against foci of infection has been a failure I have not the slightest hesitancy in maintaining that removal of the tonsils has no effect upon the course of a chorea or upon recurrence of the disease. During the last three or four years I have studied the effect of tonsillectomy quite carefully Fully as many children develop chorea after tonsillectomy as before, and, just as this little girl illustrates, almost all of my recurrent cases are in children previously tonsillectomized I am convinced that the tonsils have very little to do with chorea On the other hand, unquestionably fewer cardiac complications occur now than were reported in previous decades Whether the chorea itself has less cardiac complications or whether tonsillectomy has to do with the matter, it is impossible to state In view of the prevailing cam-

paign against tonsils, one is tempted to lay the diminished cardiac complications to the popularity of tonsillectomy I almost feel that tonsillitis, rheumatism, and endocarditis are a triad and that chorea is an independent entity grafted on top of these three But of course this is just clinical reasoning and it is difficult to prove Other observers in years gone by have occasionally questioned the close relationship of chorea to rheumatism Osler found such a relationship present in only 15 8 per cent of his chorea cases, and Sachs could find a satisfactory history of chorea coming on after rheumatism in only 20 of 184 cases Then again, rheumatism does not precede chorea nearly as frequently in the early years as it does in the ages between ten and fifteen In other words, accidental coincidence may play a greater rôle than many are willing to concede

In my own cases the association of chorea and rheumatism, or the alternating of one with the other, has been exceedingly infrequent And it is only in these exceptional cases that severe cardiac complications have developed It is only in rare instances of a combination of chorea and rheumatism that I have seen the severe endocarditis which is generally supposed to be the invariable complication of the chorea itself One gets the impression that the chorea has prepared the soil—to enable a possible symbiosis of some sort or other—for a true rheumatic endocarditis

In my own studies the focal infection idea has been disappointing It is difficult to establish any relationship between chorea and sinusitis or abscessed teeth For instance, the following findings are typical of a number of studies

Cultures from removed tonsils show a hemolytic streptococcus, from an abscessed tooth, Staphylococcus aureus, and from the pus of a punctured antrum, hemolytic streptococcus, non-hemolytic streptococcus, and diphtheroid bacilli In these cases blood-cultures were uniformly negative The organism usually described as the cause of the disease is a non-hemolytic streptococcus

The majority of our chorea children do not show definite foci of infections, and I must confess that in those cases showing

infected sinuses and teeth, I cannot see that the eradication of the foci has hastened the recovery Of course, all foci of infection should be cleaned up, that is considered the A B C of modern medicine But certainly patients with chorea do not seem to improve any more rapidly after these minor surgical procedures than they do from simple rest in bed

Another observation which makes me question the relationship of chorea to rheumatism is that I have seen a number of chorea patients get relapses or become much worse during the hot weather In my experience there may be a slight increase in frequency of the disease at the end of a summer Ordinary rheumatic fever does not come on so frequently in the hot weather Again, chorea occurs very much more frequently in girls—about 80 per cent in my cases Rheumatism shows no such preference.

And lastly we have certain circumstantial evidence in the unconvincing results of the antirheumatic streptococcic sera

I am beginning to wonder if there may not be two types of chorea Almost all observers are agreed that chorea is an infectious process closely allied to rheumatism, but in addition there seems to be a type which I have been seeing during the last three to four years, namely, a chorea caused apparently by nerve shock or nerve exhaustion in over-stimulated, overworked, and mentally fatigued children Whether these two forms have a common etiology or whether there are really two types of chorea remains to be seen

Pathology —One must say a word on this subject, although the inability to obtain pathologic material in any large number of chorea cases makes the problem complicated However, as encephalitis is so frequently followed by choreiform manifestations and as encephalitis has a fairly well known pathology, it is more than likely that any changes in chorea (if the chorea is on an infectious basis) are of the same nature, namely, in the corpora striata.

I cannot end this discussion without telling a somewhat amusing story on myself You remember that in these last years we have gotten away from the routine use of any medication because we found equally good results from simple rest in bed

A child came to my clinic some time ago with a typical chorea I sent him to the hospital where he was in bed for five or six weeks Shortly after discharge he had a recurrence Needless to say all foci of infection had been removed during the hospital sojourn In this case the father was a very nervous and rather ignorant man, utterly unable to restrain the boy from roaming the streets and visiting the movies every night I felt that the environment in this case was unfavorable and so urged another hospital stay, mainly to get the child away from his father In the hospital the youngster quieted down but upon discharge again developed symptoms, and gave the impression of becoming one of those unusual types of chronic chorea I became quite insistent that the boy be taken away from his father The patient then disappeared for some months Some weeks ago a perfectly well little boy walked into my clinic and proudly exhibited himself He was still living with his father and apparently in much the same environment, but he had not the slightest trace of a symptom He told me that for some months he had followed the advice of a neighbor and had been taking a drug-store St Vitus dance remedy I was greatly interested in learning what this marvelous cure could be and asked him to bring some He did, and I sent a specimen to the American Medical Association for chemical analysis Can you imagine my surprise and chagrin when the answer came back—"arsenic" They had not had sufficient time to determine the exact chemical combination, but inasmuch as the medicine was to be given in drops, I presume it must have been Fowler's solution So, after these fifteen years, I begin to feel that we are back where we started

Conclusions—Now I have told you all that I know about chorea and I am afraid that it is not very much Every study so far has been inconclusive There is much evidence in favor of the infectious origin of the disease, but the disappointing results of the sera have not given final proof to the hypothesis While clinical evidence always has favored the rheumatic origin of chorea, the absolute failure of tonsillectomy and the disappointing results from the treatment of other foci of infection have led me to suspect that it may not be as closely related to rheumatism

as has been thought The fact that chorea often gets worse dur ing the hot months, that it occurs overwhelmingly in girls, and that in my series the clinical association with rheumatism is most infrequent, lends some weight to this hypothesis

Again, in my cases, cardiac complications have not been severe except in those rare combinations when chorea and rheumatism have existed simultaneously Unquestionably in our series we have had fewer cardiac complications than are generally reported That this may be due to increased frequency of tonsillectomy is a possibility Tonsillectomy, however, does not prevent recurrence of chorea This rather favors the hypothesis that chorea may be a clinical entity in itself independent of the tonsillitis, endocarditis group Possibly it forms soil favorable for rheu matic infection Possibly there is a symbiotic relationship between the two conditions

In the treatment nothing can be offered but a prolonged period of mental and physical rest in bed, a period of four to six weeks The patient should not be allowed out of bed until the symptoms have entirely disappeared and until they remain absent even when the child is subjected to some mental strain

In certain cases not easily differentiated from the others arsenic may prove of considerable value

CLINIC OF DR LEROY HENDRICK SLOAN

ILLINOIS CENTRAL HOSPITAL

PERNICIOUS ANEMIA WITH SUBACUTE COMBINED DEGENERATION A PATIENT PRESENTING THE ABOVE PICTURE WITH A HISTORY OF LUETIC ENDARTERITIS AND BLEEDING HEMORRHOIDS

CHARLES K., who is fifty seven enters the hospital complaining of difficulty in walking weakness depressed sensation in abdomen soreness of the tongue, numbness and tingling in the hands and feet, and decrease in his ability to remember recent events

When this patient was twenty-one he had a primary syphilitic lesion for which he received local treatment and treatment by mouth At the age of forty-seven he had a left hemiplegia which came on one morning just as he was attempting to get out of bed At this time his blood was reported as showing a four plus positive Wassermann and his spinal fluid was also positive. Shortly thereafter he began intensive treatment by intravenous injections of salvarsan intramuscular mercury and iodids by mouth That he was adequately treated seems certain for he states that the blood tests and spinal tests have been repeatedly negative for some time. He was divorced from his wife by reason of the above events and entered on a period of vitamin starvation He would eat at restaurants. His diet was almost entirely carbohydrate with a minimum of protein and fat. He never drank milk practically never took fruit and only a very little vegetable with no butter Coffee rolls, potatoes, pastry doughnuts, and the like made up his meals. In spite of such an unbalanced diet he maintained his weight and was apparently quite well until about a year ago when the above symptoms began

At the age of fifty three he had an influenzal infection with a bronchopneumonia recovering promptly and returning to work as an insurance broker

One year ago he began to notice a general tiredness and weakness He was sleepy—would come home from his work, eat a bite and go to bed immediately He felt this weakness particularly in the legs and whereas he formerly used a cane for moral support he now needed it for physical support Numbness and tingling began to appear in the tips of his fingers and toes. He had difficulty separating sheets of paper and recognizing coins There was a band like sensation about the abdomen and slightly increased urgency of urination. Unsteadiness in walking became increasingly apparent Soreness of the tongue has been present for the last two or three months. For a

short time during this past year he visited in St Louis where he was treated for central nervous system lues in spite of negative tests

Physical Examination—A fairly well-nourished adult with a typical lemon-yellow tint to the skin who is unable to walk without support and has a tendency to fall to the left The pupils react to light and accommodation, are regular and equal The tongue is smooth, red, and shows an atrophy of the papillæ Heart and lungs are normal The spleen is at the costal margin, as is also the liver There are no abnormal masses palpable in the abdomen There are two hemorrhoids present which bleed on pressure The prostate is normal There is subjective numbness of the ends of the fingers which are reddened and pitted This in spite of the general anemia On examination of the nervous system we find evidence of an old residual hemiplegia on the left with loss of motor power to a relatively slight degree There is bilateral reduction in vibration sense, the sense of position, and muscle-joint sense in both *upper* extremities *The sensation of touch is preserved* The reflexes are active The abdominal reflexes are present but reduced There is a very marked reduction in the muscle-joint sense, tendon sense, and vibration sense in both *lower* extremities and while there is slight reduction in the sense of touch it is almost negligible *The knee-jerks are double plus on both sides, the Achilles jerks also greatly exaggerated and there is an extensor response on stroking the sole Ankle clonus is present in both ankles* There is marked loss of motor power in both legs and feet Ataxia is present *The finger-to-nose, finger-to-finger, heel-to-knee, toe-to-finger tests are poorly performed* The *Rhomberg* test is positive with more tendency to fall to the left which is due to the old hemiplegia The *spinal fluid is entirely negative* and the *blood Wassermann* is also absolutely *negative* The urine shows a slight trace of albumin from time to time

When we examine the blood we are confronted with the following Hemoglobin 60 per cent , erythrocytes 2,600,000, leukocytes 8700 with anisocytosis, poikilocytosis, polychromatophilia, increased macrocytes, and an occasional nucleated red cell The stool shows no blood within the substance but occasional streak of blood on the external surface—due to the hemorrhoids However, we are told by the patient that there have been several occasions when the stool showed much red blood

Discussion—What are we dealing with in this patient? We feel certain that the old hemiplegia was due to a syphilitic endarteritis with thrombosis Is the present condition due to syphilis? Is this tabes? No—it is not Why? First, the pupils are perfectly regular and react promptly This finding is important Second, the tongue is not that of syphilis, it is absolutely typical of pernicious anemia Thirdly, we find an *exaggeration* of the patellar and Achilles reflexes with *clonus* and *extensor response* The sense of touch is well preserved This is not tabes Tabes shows reduction in the reflexes with flexor response, and delayed

sensation Further, the blood and spinal fluid are negative And again, we have a blood picture which is typical of pernicious anemia What one added finding would convince us that this is pernicious anemia with cord changes? *The gastric analysis* Now we find on both ordinary Ewald meal and on fractional tests of the gastric content an absolute achylia Also of importance here is the absence of pain This patient complains of numbness and tingling but there is no real pain

Discussion of the Etiology —I believe that we are allowed to put aside the syphilitic factor as causative of the condition under

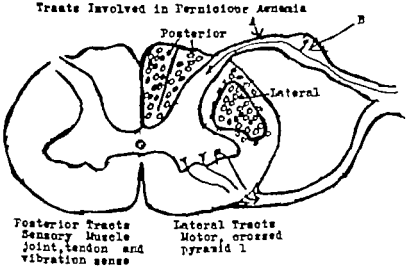

Fig 75

discussion More easily can we do this in view of adequate treatment and negative findings What other factors are possible? Two Long-continued faulty diet with a preponderance of carbohydrate and an almost total lack of vitamin containing foods might explain it Bleeding hemorrhoids will give rise to a picture closely resembling that of pernicious anemia, and while we have such a history here it does not seem possible to believe this one factor to be sufficient We do know that a picture of cord changes may be produced in experimental animals by prolonged bleeding

Subsequent History —This patient was transfused several times, put upon added diet, and given hydrochloric acid and pepsin He improved slightly The hemorrhoids were removed

Iron and arsenic were given hypodermically With the advent of the specific diet of Koessler and his co-workers this patient was returned to the hospital, put to bed, and treated according to the ideas of the late Karl Koessler His improvement began at once Within a relatively short time the blood hemoglobin has risen to 90 per cent and the red cells to over 4,500,000 Weakness became less marked, tiredness disappeared, and there was an apparent reduction in the ataxia and muscle weakness On examination in December, 1928 this patient still has his numbness and tingling, the pitting of the finger-tips is still present, but he is back to his occupation and carrying on Reflexes, clonus, reduction in muscle, joint, tendon, and vibration sense still remain as on the first examination but the ataxia has been much lessened He is still taking liver extract but has stopped other medication His blood-count shows a hemoglobin of 90 per cent with 4,300,000 red cells The achylia is still present

ACOUSTIC NEUROMA

R. W. age twenty years was referred by Dr. W. F. Hewitt with the following history:

Eighteen years ago he fell since which time he has had a left convergent strabismus. One year ago had a series of boils and carbuncles. Ten months ago began to stagger. Tended to go toward the left. Friends thought him drunk. Noticed that his left leg doubled under him. Six months ago had a spinal puncture following which he had a severe headache for eight days. About this time could not walk alone. Recovered somewhat and is now able to walk alone but with a wide base and with a tendency to fall to the left. He has been very sleepy for two or three months. About one month ago noted rapid reduction in hearing on left. For the past two weeks has had difficulty in making out objects. Persons coming up on the left are not appreciated so he states. His left arm has been unsteady. Has a tired sensation between the hip and nipple area on the left. For a short time he has had some slight difficulty in closing his left eye. There have been no convulsions. There have been attacks when everything would go black before him.

Examination this morning shows the following important findings:

Pupils react to light and to accommodation are regular and equal. No gross defect in the visual fields found. Nystagmus is present with the rapid component to the right and the slower larger component to the left. Vision 20/50 in both eyes. There is a bilateral papilledema of 2 diopters. The corneal reflex is definitely reduced on the left. An old external rectus paresis is found on the left. Hearing in the left ear is greatly reduced. The muscles of the left face are paretic. The action of the left palate is reduced. The abdominal, patellar, plantar, Achilles reflexes are normal. There is ataxia, dysmetria and hypotonia of the left arm and left leg as noted in the finger to-nose, finger to-finger, heel to knee, heel to-shin and toe-to-finger tests. The Romberg is positive with falling toward the left and backward. No sensory changes are noted.

The blood-count is normal, the spinal fluid under much increase of pressure but showing no abnormal findings. The urine shows a trace of albumin and a trace of sugar. A stereo plate of the skull is negative.

Discussion.—Now what do all these findings mean and how shall we interpret them? The nystagmus is quite characteristic of cerebellar involvement. It is suggestive of a left cerebellar involvement. The bilateral papilledema means but one thing—increased intracranial pressure especially in the absence of renal changes and hypertension which sometimes confuse. What of

the corneal reflex? This means involvement of the fifth cranial nerve What does the paresis of the external rectus mean? Nothing so far as the present condition is concerned This is an old trouble The paresis of the muscles of the face with difficulty in closing the eye means a rather extensive involvement of the seventh nerve on the left The loss of hearing of course means that the eighth nerve is involved What do the ataxia and dysmetria and hypotonia of the left arm and leg mean? Further involvement of the cerebellum or its important connections The Romberg with falling toward the left and especially backward means the same thing The patient compensates for many of his findings by walking with a wide base When he is suddenly pushed backward he falls If pushed to the left he falls also When asked to stand with eyes closed he falls immediately backward and to the left

Interpretation—With headache, papilledema, and a progressive increase in the symptoms and signs the most likely diagnosis is that of brain tumor Where is this tumor and what is it? Our findings point to one spot—the cerebellopontine angle Why? Because it is at this point that the eighth nerve, the seventh nerve, and the cerebellum are in intimate contact At this point, also, all of the findings above recorded may be accounted for The reduction in the corneal reflex on the left only confirms our impression of such a location Now what is the most common lesion in this location? A neoplasm arising from the acoustic nerve Through growth and compression a tumor of this nerve injures the seventh or facial nerve and irritates or injures the cerebellum Ordinarily in such a tumor in this location we find an increase in the cell-count and in the protein of the spinal fluid We do not in this case Again, it is not a wise plan to puncture the spinal canal of patients showing these findings We did so here because of a previous suggestive spinal Wassermann and a history in the family pointing to lues as a possible etiologic factor The puncture was done with the patient in bed, head down and feet up

Subsequent History—This patient was operated upon by Dr Percival Bailey and a primary acoustic tumor removed

CLINIC OF DR CLARK W FINNERUD

COOK COUNTY HOSPITAL

DERMATOLOGIC CLINIC

LEIOMYOMA CUTIS

THIS man, aged sixty five years, foreign born has a cutaneous disorder of the back, just below and lateral to the right scapula, of indefinite duration He is referred to this clinic from one of the surgical wards where he is under treatment for a surgical condi tion He has made no complaint about the skin disorder, it merely being discovered during the routine physical examination

Examination reveals a hand-sized area composed of discrete brownish red, firm, oval nodules, which vary in size from that of a match head to that of a coffee bean most of them being of the latter size There are several dozen of these nodules in the group, and they are elevated from 1 to 3 mm above the surrounding skin Apparently there are subjective symptoms present in the patch, in that on manipulation of the lesions the patient cringes and tries to draw away from the examining hand

Leiomyoma is the name given to the tumors which are com posed solely of smooth muscle fibers, one of the rarest of the cutaneous tumors Clinically, its characteristics are so uniform that usually it can be recognized at a glance, although one of the nodules should always be excised for histologic examination by way of removing all doubt as to the diagnosis There are only about 50 cases of this disorder on record and these were recently summarized by Dr Ormsby (see Archives of Dermatology and Syphilology, 1925, xi, 466) Within the last five years there have been 5 such cases presented before the Chicago Dermatological Society and none prior to that date

The lesions of leiomyoma cutis occur in hand-sized or smaller patches, chiefly on the face, trunk, and upper extremities There is usually but one area of involvement Subjective symptoms may be absent or the lesions may be sensitive to pressure and changes in atmospheric temperature Not uncommonly there are paroxysms of pain in the area A patch is made up of numerous discrete lesions, these sometimes being so closely set as to form plaques The individual nodules average the size of a split pea and are usually oval, although some of them are round The oval ones for the most part have their long axes approximately parallel to each other The lesions may be dull red, yellowish red, reddish brown, or with a bluish tinge

Leiomyoma cutis is of unknown etiology It occurs more commonly in the male and is essentially a disorder of adult life Half of the reported cases have begun before the twentieth year, however Histologically one sees a tumor mass occupying the greater portion of the corium This mass is composed of bundles of smooth muscle-fibers which course in all directions, some of them being cut longitudinally and some transversely or obliquely The amount of fibrous tissue around these bundles is variable but in some instances this fibrous element is so markedly increased that it is necessary from a histologic standpoint to consider the tumors fibroleiomyomata instead of pure leiomyomata Cutaneous smooth muscle tumors usually originate from the arrectores pilorum, but sometimes from the muscularis of the blood-vessels

Leiomyoma cutis may resemble syringocystoma clinically, but in the latter disorder the lesions are symmetrically disposed and the nodules are soft In such and all other instances the diagnosis is immediately settled histologically There is no very satisfactory treatment for leiomyoma cutis, although often it is practicable, especially in the instance of a small patch, to remove it surgically Freezing with carbon dioxid snow removed some of the lesions in the cases treated by Dr Ormsby and relieved the pain in all Roentgen therapy seems to be of little, if any, service in the management of this disorder

FIXED EXANTHEM (PHENOLPHTHALEIN ERUPTION)

This woman, aged thirty-six years, has a recurrent eruption of the flexor surface of the forearms, right buttock, and medial surface of the thighs It was first noticed about a year ago and since that time she has had about a dozen attacks each lasting from two to seven days The present attack is of one day duration She states that the lesions always recur in exactly the same spots, as red, slightly itching areas, and that after the redness has disappeared, there remains a brownish discoloration which persists until the beginning of the next attack She took two tablets called "Nature's Remedy" the night before the present lesions were noticed

This form of *dermatitis medicamentosa* is usually spoken of as the "fixed exanthem" or "fixed eruption " It is usually produced by the ingestion of phenolphthalein, a common ingredient of proprietary laxatives The eruption appears within a few hours after the taking of such a laxative in any individual who is properly sensitized to that drug Usually from the history of the case, it can be determined that phenolphthalein is the cause Occasionally it is necessary to prove that this drug is or is not responsible for the eruption by administering it to the patient while he is in the quiescent stage The same type of eruption sometimes is produced by the ingestion of antipyrin or arsphenamin, and more rarely by certain other drugs

Active lesions occur as round, coin-sized or larger, bright red to bluish red macules, each sharply demarcated from the surrounding skin There are usually rather mild itching and burning sensations in these lesions The red macules appear a few hours after the ingestion of the drug Within three or four days the redness disappears and is replaced by a light or dark brown macule of exactly the same shape and size This brown macule persists until the time the drug is next taken, at which time it becomes an erythematous macule again This is the most common eruption due to the ingestion of phenolphthalein, although this drug is sometimes responsible for a vesicular and crusting dermatitis with superficial ulceration of the lips, mouth, and genitalia

An individual so afflicted should simply be instructed to a proprietary laxatives and cathartics, and in case such an age necessary, to use a preparation known to contain none of drug

PSEUDOPELLAGRA

A negress, aged thirty-two years, born and raised in Chic complains of loss of weight, increasing weakness, diarr nervousness, sore tongue and mouth, and inflammation of skin of the back of the hands and distal third of forearms, including the flexor surface of the wrists, of appr mately two weeks' duration Upon questioning her further learn that she has been eating practically nothing for about weeks, but that during this time has consumed from a pin more than a quart of "moonshine" daily, liquor which she bou for 60 cents per quart Her personal, past, and family histories without noteworthy features

Examination reveals an emaciated woman, so weak that can scarcely rise alone from the wheelcart Her speech is rat incoherent and there is tremor in her hands Her tongue has appearance of "raw beefsteak" There is an erythemat scaling and crusting dermatitis, moderately infiltrated a sharply demarcated, of the back of the hands and distal third the forearms The palms are unaffected, but a "gauntlet" formed about the forearms

In the past few years cases of this type have been seen increasing frequency here at the Cook County Hospital Th present all of the classical features of true pellagra, but becau of the invariable history of long-continued drinking of alcoho preparations, practically to the exclusion of all other nourishme the condition has been termed *alcoholic pellagra* or *pseud pellagra* These cases run essentially the same course as tr pellagra and clinically cannot be differentiated from it The are probably a dozen or more of these patients admitted to th institution annually The disease is seen in all grades of severit sometimes merely a sharply demarcated, erythematous or bullo dermatitis being present on the back of the hands, associated wi diarrhea and a mildly inflammatory tongue and mouth lining,

there may be a more extensive pellagrous cutaneous eruption associated with severe diarrhea, marked ulcerative changes in the mouth and alarming degrees of emaciation, weakness, and delirium Incidentally, you all remember the 'three D's" as among the cardinal symptoms of pellagra, namely, dermatitis, diarrhea, and delirium It is hardly expedient at this time to detail the extensive neurologic and other symptoms which may be present in pseudopellagra, as they are identical with those previously discussed under pellagra

Concerning treatment, it may be said that the patients who enter the hospital in a moribund state seldom recover Those who will eat most or all of the food placed before them invariably get well We have had greatest success with the diet composed of red meats, green vegetables, butter, milk and cream, three yeast cakes daily, and the juice of citrous fruits It is remarkable the rapidity with which these patients recover, usually almost completely regaining their strength and weight and losing all of their symptoms within two to four weeks A simple protective paste, such as the zinc oxid ointment or Lassar's paste, ordinarily is all that is necessary in the way of topical cutaneous preparations A well-diluted Dobell's solution may be used to advantage as a mouth wash three or four times daily

IMPETIGO CONTAGIOSA

A man, aged thirty-four years, has an eruption of the bearded region of six days' duration He believes that it was contracted in a barber shop, as it began the day after he was shaved by a barber He states that this was the first time he had been shaved by anyone except himself for many months There are practically no subjective symptoms He has been using some proprietary remedies suggested by a druggist without improvement, preparations which the druggist said were good for "barber's itch"

Upon examination you will see numerous, fingernail sized and larger, serous-crusted plaques of the bearded portion of the chin and cheeks, many of these crusts having a "stuck-on"

appearance Some of the lesions are discoid and others are circinate

Impetigo contagiosa is a disorder essentially of children, and if one child of a family has it, usually several or all are affected It is not a contagious disorder, as the name implies, but it is highly infectious The lesions occur for the most part on the exposed surfaces of the body, notably the face and hands They usually exist as finger-nail sized and larger, serous-crusted areas, circinate lesions being unusual The crust usually is definitely larger than the underlying inflamed area, that is, it overlaps the true lesion

In infants this eruption is not uncommonly a bullous one and is, therefore, properly spoken of as *bullous impetigo* and improperly referred to as "pemphigus neonatorum" The latter name is to be condemned, in that the disorder is in no way to be considered a form of pemphigus Bullous impetigo not uncommonly terminates fatally

When impetigo occurs in adults, it is usually in the male and practically always the result of barber-shop transmission When present in women it is usually contracted from children If untreated, or mistreated, it tends to spread rapidly It is probably never with serious consequences in adults

A man so afflicted is usually told by the barber and by his friends that he has "barber's itch" This, of course, is incorrect, true barber's itch, more properly referred to as ringworm of the beard or *hyphogenous sycosis*, being one of the rare cutaneous disorders In ringworm of the beard itching is a prominent symptom and the ringworm fungi can be readily demonstrated in the hairs and scales in KOH and NaOH fresh preparations It should be mentioned that impetigo is not to be confused with the recurrent extralabial form of herpes, that is, herpes simplex In the latter disorder there is usually a history of "cold-sores" being present following exposure to sun and wind or at the time of ordinary colds or any illness which might be responsible for fever Herpes simplex occurs as one or more split-pea sized or larger groups of vesicles, which may or may not tend to rupture spontaneously Vesicles are practically never demonstrable in

impetigo, the lesion in the latter disorder being a weeping area or crust almost from the time of its inception

Impetigo contagiosa is due to bacteria, streptococci chiefly, which presumably are present to some extent normally on the skin of all of us They gain access into the skin at the site of an abrasion, such as might occur from a scratch, a cut, or picking the face with dirty finger nails, or the disorder may occur secondarily to a "running" nose or ear It spreads rapidly, several or many lesions being present within a few days after the disorder is contracted It may be transmitted from one individual to another from the use of a common towel or washcloth and by all forms of intimate contact

The mercury group, properly employed, is the treatment of choice in cases of impetigo The patient should be instructed to bathe the parts morning and evening for about ten minutes with a warm solution of mercury bichlorid, this varying from 1 2000 to 1 5000 in strength Following the bathing in the morning he should apply a calomel dusting powder of about 12 per cent. strength, this to be left on during the day The formula for such a powder would be Mild chlorid of mercury, 4 grams, boric acid, 4 grams, and equal parts of starch and talc to make 30 grams Following the bathing at night, an ointment containing from 3 to 6 per cent of white precipitate should be used, this to be left on over night In case the inflammatory element is particularly marked, which is not usually the case, it is best to combine the white precipitate with a naftalan ointment of 10 per cent strength The formula for such an ointment would be Ammoniated mercury, 1 gram, naftalan, 3 grams, starch and zinc oxid, each 8 grams, and petrolatum sufficient to make 30 grams

In that there is perhaps one individual in a thousand who is hypersensitive to mercury preparations, in his particular instance it is necessary to stop the mercurial preparations and to use such a treatment as potassium permanganate bathing of the parts followed by rather mild sulphur preparations

The above measures will manage practically any case of impetigo within five to ten days it rarely being necessary for the individual to make a second visit

MOLLUSCUM CONTAGIOSUM

A girl, aged seven years, complains of an eruption of the face, trunk, and extremities of about six weeks' duration The mother states that the lesions look somewhat like warts to her and that they are increasing in size and number rather rapidly The first lesion was noticed on the left cheek and this was picked by the patient to the extent of making it bleed This particular lesion has disappeared, but it is the mother's impression that no others have done so There are no subjective symptoms

Molluscum contagiosum is seen chiefly in children It may be spread from one child to another by intimate contact Although not a contagious disorder, it is definitely infectious The organism responsible for it has not been isolated, but it is presumed to be a filtrable virus

The lesions vary in number from one or two to a hundred or more and are pinhead- to match-head sized or larger, waxy-looking, firm, flat, round nodules In the center of each lesion is a small depression which grossly appears to be filled with a lusterless débris This central portion sometimes presents the appearance of the surface of a small wart of the ordinary type and usually it can be easily expressed

The disorder is so characteristic clinically that histologic examination is not necessary However, the microscopic picture is a constant one and entirely different from that of all other epithelial tumors

The lesions are best treated by transfixing their bases with a flat surgical needle This causes slight hemorrhage in the lesions and is sufficient reaction to bring about their disappearance within a week or ten days No scarring results from this method of treatment, its only drawback being that it is somewhat painful

BLASTOMYCOSIS

This man, aged thirty-seven years, a stockyard worker, has an eruption of the face of three months' duration He states that the lesions began as pimples which became warty and gradually enlarged to their present size There are no subjective symptoms

Upon examination are seen a half-dollar sized crusted plaque on the front of the right cheek, its margin of somewhat verrucous appearance and bluish red, and a similar but smaller lesion of the right temple and one of the side of the right cheek The border of the lesions slopes abruptly and there is elevation of about three eighths of an inch in each On the borders are seen an occasional pinhead-sized or smaller pustule, lesions spoken of as miliary abscesses Under the microscope you will see budding yeasts, blastomycetes, in a 20 per cent sodium hydroxid preparation from one of these minute abscesses

Blastomycosis is seen with frequency in the Chicago area and a large proportion of these cases first present themselves here at the Cook County Hospital It is a chronic inflammatory disease produced by an yeast, the blastomyces

The majority of the cases are purely cutaneous ones, but in rare instances the disorder becomes systemic Usually there are but two or three skin lesions present, although there may be a couple of dozen or more The lesions begin as bluish-red papulopustules, usually on the face, which slowly grow to attain the size of a coin or larger Well-developed lesions exist as bluish-red, papillomatous, flat nodules, well elevated from the surrounding skin, the center of each of which is a superficial crusted ulcer The base of this ulcer is usually above the level of the surrounding normal skin The lesion is usually with an elevation of about ¼ to ½ inch and its margin is steep and stippled with minute "miliary abscesses" It is from these abscesses that the yeast responsible for the disorder can be readily demonstrated

In cases of systemic infection from this organism the cutaneous lesions usually have entirely different clinical characteristics from the lesions of external origin In the systemic cases there occur crops of cutaneous and subcutaneous nodules and abscesses, hazelnut to hen's egg sized or larger, associated with constitutional symptoms These nodules often soften, fluctuate, and break down, thus forming large, irregular, crusted ulcers with a soft but granulating floor

There is practically no tendency toward spontaneous cure in blastomycosis, but the disorder responds rapidly to the proper

therapy, healing with a coarse, pinkish or whitish scar Spontaneous healing does often occur, however, in the center of large lesions, the periphery remaining active

The blastomyces is readily demonstrated in potassium or sodium hydroxid preparations of the pus and slowly demonstrated by culture or the finding of it in histologic sections The glairy, slightly turbid, mucus-like content of the miliary pustules on the sloping border of the cutaneous lesions contains the organisms in abundance In fresh preparations they exist as doubly contoured, highly refractile, round or budding bodies with a slightly granular cytoplasm They are usually about 20 microns in greatest dimension A positive diagnosis cannot be made from the presence of round forms alone, it being necessary to demonstrate bottle-shaped or other budding forms before such a diagnosis can be made This yeast grows on practically all ordinary media at room temperature, the colonies being definite within less than a week's time

Blastomycosis, histologically, may assume much the same appearance as tuberculosis verrucosa cutis or, in rare instances, squamous-cell epithelioma Ordinarily, however, there is no doubt concerning the histologic picture, even in the absence of demonstrable organisms in the section

Clinically, blastomycosis is most likely to be confused with a granuloma produced by the ingestion of bromid, squamous-cell epithelioma, and tuberculosis verrucosa cutis A positive diagnosis rests upon the demonstration of the blastomyces, usually a very simple matter and one requiring but a few minutes' time With inability to demonstrate the organisms one should immediately suspect bromoderma and inquire of the patient as to long-continued taking of sedatives, nerve tonics, etc It may be necessary to perform a biopsy by way of differentiation of this disorder from the squamous- or prickle-celled cutaneous cancer, but this would be an extremely exceptional instance

In all except the advanced systemic cases the disorder responds rapidly to potassium iodid taken in increasing dosage up to as much as 150 to 300 drops of a saturated solution daily The treatment should be reinforced by the intrave-

nous injection of arsphenamin and the use of a 3 to 5 per cent ammoniated mercury ointment and proper Roentgen ray exposures locally

PSORIASIS (LIMITED TO THE NAILS AND SCALP)

A man, aged forty six years, complains of a disorder of the finger nails of about ten years' duration He states that the condition gets better and worse, but that he believes he has never been entirely free from it since the onset The nails are always at their worst in cold weather He states that he has had no skin disorder, but upon further questioning admits that he is troubled with considerable dandruff like scaling of the scalp, particularly in winter He has had no illnesses of any consequence and his family history is essentially negative, aside from the fact that one of his brothers has a scaling eruption of the scalp, trunk, and extremities of many years' duration

Upon examination there are seen numerous punctate depressions of the surface of the finger nails, and these nails are yellowish and thickened at their free margins, especially at the corners This yellowish undermining process involves approximately the distal third of each nail The toe nails are grossly normal, except for this same type of undermining and thickening in the distal half of each great toe-nail There are a half dozen or more coin sized patches of dense scaling in the scalp When the scales are removed there is exposed a sharply demarcated, infiltrated, dry, reddish plaque Upon curetting off the scales an occasional minute bleeding point is seen He has a good head of hair, and there is no evidence of other cutaneous disease

Psoriasis of the nails, well illustrated in this case, usually is of a characteristic clinical appearance The surface of some or all of the nails, particularly those of the fingers, is stippled with from two or three to twenty or more punctate depressions which give it more or less the appearance of the surface of a thimble Psoriasis also affects the nails as an undermining and thickening, beginning at the corners distally and progressing toward the center of the nail and backward, thus loosening the nail from its bed in its distal portion and giving it a "clouded amber" or

even "worm-eaten" appearance The pitting type of involvement or the undermining type may occur separately or, as in this case, they may occur together Probably in less than one-fourth of the cases of psoriasis is there nail involvement and, when present, it is usually associated with rather marked cutaneous manifestations of the disease

The favorite sites of psoriasis are the scalp, elbows, knees, and extensor surfaces in general It rarely affects the exposed surfaces, such as the face, neck, and hands, and never the mucous membranes Psoriasis is characterized by well-infiltrated papules and plaques, which may be punctate, guttate, nummular, or of large plaque type Annular lesions and serpiginous eruptions are not uncommon Upon undergoing involution the lesions tend to clear primarily from their centers The scale of psoriasis is a silvery white or "mother of pearl" colored, imbricated one which is laid down in layers, more or less like the shingles on a roof The lesions usually are without subjective symptoms Psoriasis does not cause alopecia Individuals with psoriasis usually have a subnormal blood-pressure

The cause of psoriasis is unknown, although there are many theories concerning its etiology There is some evidence for the belief that it is the result of disturbance of balance of the nitrogen metabolism and there is also fairly sound evidence in support of the theory that it is of infectious origin Its occurrence in more than one generation of a family or in more than one member of the same generation is so common that it seems possible that this is more than coincidental, in other words, that possibly there are hereditary factors It is one of the most common of skin disorders By far the majority of the cases are worse in cold weather

So far as the differential diagnosis of this particular case is concerned, the scalp lesions need only be differentiated from seborrheic dermatitis In the latter disorder the scales are yellowish and greasy and the lesions are not so sharply defined Upon removing the scales in seborrheic dermatitis a moist surface is exposed Upon removing the lowermost scales in a lesion of psoriasis there appear minute bleeding points In

seborrheic dermatitis usually there is periodic itching Psoriasis of the nails is most likely to be confused with ringworm of the nails. The latter disorder begins at the distal end of the nail, usually undermining it in much the same way as may psoriasis, thus giving the nail a similar appearance to that of the undermining type of psoriasis Scrapings of the under surface of the nail show ringworm fungi in potassium or sodium hydroxid fresh preparations if ringworm is present In the absence of such findings one may be quite sure that he is dealing with psoriasis, even in the absence of the pitting type of involvement Any disorder which causes inflammation about the base of the nail, such as eczema or paronychia, produces dystrophic changes in the nail which begin at the proximal end In the instance of syphilis we have a disorder which may affect either the distal or the proximal end of the nail primarily

Psoriasis of the scalp is best treated by nightly applications of a 15 per cent white precipitate ointment To this might well be incorporated 2 or 3 per cent of salicylic acid Psoriasis of the nails responds best to borax bathing followed by nightly applications of a 3 to 10 per cent salicylic acid ointment. The nails should be kept closely filed Arsenic internally is of definite service in these chronic cases You are all aware of the dangers of prolonged arsenic taking and it need, therefore, scarcely be mentioned that an individual taking it should do so only while under the observation of his physician The nail cases are particularly resistant to treatment and respond rapidly only to Roentgen ray exposures It should be remembered that x-ray treatments should be given with great caution and only by one skilled in the use of this agent in the treatment of skin diseases For reasons apparent, this form of therapy should be resorted to only as a last measure.

PITYRIASIS ROSEA

This young woman, a blonde, complains of a breaking out of the trunk, arms, and thighs of two days' duration She states that she is feeling well and that there are no subjective symptoms She has had no previous skin disease aside from an occasional

pimple of the face and shoulders, a condition which is usually present only at the time of her menstrual periods

Upon examination there are seen numerous maculopapules, both round and oval, of all surfaces of the trunk, and of the thighs and arms By far the largest lesion is this silver-dollar sized one over the left scapula The others average the size of finger-nails There is no superficial lymphadenopathy and the mouth is of normal appearance

Pityriasis rosea usually begins with a lesion called the "mother patch" or "herald patch," this lesion being the largest and most highly colored one present and preceding the more extensive eruption by four to ten days This herald patch is demonstrable in about 75 per cent of the cases and is often so situated that it is overlooked by the patient Probably its most common site is the side of the trunk The subsequent outbreak is characterized by lesions such as you see here, round and oval macules and maculopapules, with scaling, clearing, and slightly depressed chamois-colored centers The border of each is more distinctly red and slightly elevated, giving the lesions somewhat of an annular appearance An outstanding characteristic of this eruption is the fact that the oval lesions are all so directed that their long axes are parallel to the lines of cleavage of the skin

Ordinarily there are no prodromata, but in some instances there are mild fever, headache, malaise, and other general symptoms The disorder seldom extends above the collar line or below the elbows or knees In the beginning there may be associated a mild superficial lymphadenopathy Pityriasis rosea runs its course, as a rule, in from three to eight weeks, and there are no sequelæ There are but a few instances on record of recurrence, which leads us to believe that it must be of infectious origin and capable of establishing an immunity

The cause of pityriasis rosea is not known, but it presumably is, as stated, of infectious origin It occurs chiefly in the spring and fall, and, though not a common disorder, is sometimes seen in almost epidemic proportions It is probably more common in women than in men and most of the cases occur in the spring

and fall Blondes are more commonly affected than others, and it is essentially a disorder of adults under middle life

Pityriasis rosea is most likely to be confused with the maculopapular eruption of secondary syphilis, seborrheic dermatitis, psoriasis, drug eruptions, and ringworm In the instance of secondary syphilis the concomitant symptoms of that disorder may be expected to be present at least in part Although the lesions of seborrheic dermatitis of the body may closely simulate in appearance those of pityriasis rosea, in the former disorder the sites of predilection are a V of the chest in front and behind, associated with greasy scaling of the scalp, eyebrows, and ears Itching, usually absent in pityriasis rosea, sometimes is a distressing symptom in seborrheic dermatitis Drug eruptions usually can be ruled out by history taking, and the lesions of psoriasis, when small, practically never have clearing centers, but a silvery imbricated scale, in contrast to the furfuraceous one of pityriasis rosea In ringworm the fungi can be demonstrated readily in the scales

Treatment is hardly necessary in pityriasis rosea, although a mild keratolytic ointment seems to hasten its course Such an ointment might contain 1 per cent of salicylic acid and 2 per cent. of sulphur put up in equal parts of cold cream and vaselin this to be applied each night upon retiring

CLINIC OF DR JACOB MEYER

MICHAEL REESE AND COOK COUNTY HOSPITALS

DIAGNOSIS OF PEPTIC ULCER

In a previous clinic I illustrated the essentials in the diagnosis of typical cases of peptic ulcer These essentials may be summarized as follows

1 A careful analysis of the *history*, which discloses a story of epigastric distress recurring regularly a half hour or longer after meals, and relieved by soft foods and alkalis Such discomfort occurs not only daily after meals, but at intervals of three to six months This condition repeats itself for a number of years so that the terms "periodicity" and "chronicity" are applied to the picture of peptic ulcer

2 *Roentgenographic* evidence (*a*) Direct, such as persistent deformity of the lesser curvature, the so called "Haudek's niche," retention of barium, defective duodenal cap or persistent deformity of the duodenum, (*b*) indirect, persistent spasm, hyperperistalsis, hypersecretion

3 A localized area of tenderness in the epigastrium, in the midline for gastric ulcer and 2 to 4 cm to the right in duodenal lesions This area of tenderness is best elicited by gentle percussion with a reflex hammer

4 Repeated positive tests for blood in stomach contents, especially in association with hyperacidity, but of equal importance in normal acidity or subacidity Repeated positive tests for blood in stools

Further considerations of the diagnosis of ulcer include the various complications encountered as a result of the complications These may be mentioned as gastric hemorrhage, pyloric stenosis, penetrating ulcer, perforating ulcer, ulcer with carcinomatous changes, [illegible] stomach, subphrenic abscess

In private practice you may encounter many variations of peptic ulcer syndrome which trouble not only the beginner but the most experienced

Case I.—The first case which I will relate to you is a woman aged thirty-six years, who complained of epigastric discomfort, and occasional attacks of pain in the right upper quadrant The relation of pain to food was not definite The patient described the sensation as one of fulness and heaviness Sodium bicarbonate gave relief of these symptoms She was rather excitable and inclined to exaggerate her discomfort Ewald test-meal—Free acid 20, total 45, blood ++ Feces—blood ++++ The first impression was that of a mild cholecystitis The Graham-Cole dye test was normal, and direct plates of the gall-bladder were negative Fluoroscopy and Roentgen plates (Fig 76) showed a distinct deformity of the duodenal cap This led to

Fig 76.—Duodenal defect—indicating ulcer

a diagnosis of duodenal ulcer, despite the absence of a typical story A glance at Fig 76 will emphasize that typical *x*-ray evidence of ulcer must not be ignored in the absence of typical ulcer story

Case II.—Another excellent illustration of this is the case of a young man aged twenty-eight years, admitted to the service at Cook County Hospital In 1927 the patient complained of pain in the abdomen and back which was so severe that he discontinued work for a period of a week There was no vomiting during this period, but nausea was present and the patient observed bloody stools He felt better after a week, and was advised to remain on a soft diet He has since complained of dull aching pain in the epigastrium and

[illegible] when he works is not affected by meals but he obtains [illegible] of relief by soft foods and bowel movements. He is constantly [illegible] has resorted to cathartics for relief. There were no other [illegible] which might suggest a story of ulcer. The routine [illegible] on this patient and it was shown that although the free

Fig. 77.—Defect on lesser curvature—indicating penetrating ulcer

hydrochloric acid was 19 and total acidity 50 blood was present in the stomach contents and also in stools. The *x*-ray picture (Fig. 77) completed the diagnosis showing a distinct defect on the lesser curvature, indicating a penetrating ulcer.

Lest you mistake the significance of these cases, I wish to emphasize that the *x*-ray examinations will establish the diagnosis in atypical cases, but that in the ordinary typical ulcer the history is of first importance.

You may then ask concerning that group of patients who present symptoms of definite gastric distress which may or may not be associated with hyperacidity, and which show no a-ray evidence, direct or indirect How shall these be regarded? You will also recall that there are superficial acute ulcer, hemorrhagic erosion, or multiple ulcers which may be symptomless, except for hemorrhage In general, I proceed to eliminate all the known causes for hyperacidity and its symptoms which may simulate ulcer Some of these causes are chronic cholecystitis, simple constipation, chronic appendicitis, colitis, visceroptosis, gastric neurosis, incipient pulmonary tuberculosis

Chronic Cholecystitis—The history may be identical with that of duodenal or peptic ulcer The occurrence in the female should favor the diagnosis of cholecystitis Respiratory embarrassment, variously described, should favor gall-bladder disease Belching, while present in both conditions, is more common in my experience in patients with gall-bladder disease It must be remembered that severe attacks of epigastric discomfort occur in peptic ulcer, but these attacks are generally not as severe as those periodic attacks of pain occurring in gall-bladder disease The presence of fever, leukocytosis, tenderness in the region of the gall-bladder, or enlargement of the liver at the time of attacks favor cholecystitis In the chronic cases of cholecystitis tenderness is present over the right lower costal cartilage, in contrast to pain and tenderness in the midepigastrium If a story of jaundice, or a positive van den Bergh test, or positive cholecystogram is obtained, the diagnosis is not difficult Hyperacidity may be present in acute cholecystitis as in duodenal or peptic ulcer, but the presence of blood in the stomach contents and stools should favor the diagnosis of peptic ulcer It must be remembered, however, that a small percentage of gall-bladder cases do show blood in the stools and gastric contents Finally, it must be remembered that chronic cholecystitis and duodenal ulcer may coexist

Chronic Appendicitis—The term "chronic appendicitis" is subject to much abuse I use it to convey a condition in which there has been a definite story of an acute attack with pain,

localized tenderness, fever, and subsequent recovery with recurrence of mild or moderately severe attacks resulting in a chronic inflamed or adherent appendix This may be associated with the ulcer syndrome and at times the differential diagnosis is difficult Exploratory operation may reveal the true nature of the lesion Appendectomy results in a cure in this form In contrast to this group of chronic appendicitis there is another group named "chronic appendicitis" which offers even greater difficulties Symptoms of appendicular colic may occur but they are not primarily inflammatory in origin The history often simulates a true chronic inflammatory appendicitis, but the patients present symptoms and other factors which place them in the group we term "neurotic." Thus they are easily excited, show marked vasomotor instability, complain of varied abdominal distress, belching, pain in epigastrium and on the right side, constipation, at times even mucous colitis Hyperacidity is associated Roentgenograms often show a delay in the emptying time of the appendix These patients should not come to operation Experience I believe proves that they are not improved by operation but often continue to complain They should be treated as I shall discuss in the next group

Gastric Neurosis —This is a term which some of you may be familiar with as implying disturbances in motor and secretory functions, such as hypo and hyper acidity, pylorospasm aërophagia, bulimia While these conditions undoubtedly may occur, they are only of direct concern to the practitioner inasmuch as they produce the discomfort expressed by the patient as "stomach trouble," heart burn, choking or "lump in the throat after eating" At times the story may be typical of ulcer A proper evaluation of the history and physical findings should form the basis for diagnosis In many instances the "fear" of ulcer is the cause of the ulcer syndrome A complete physical and roentgenologic examination will often convince the patient of his error It is not infrequent to find patients with social difficulties, domestic, sexual, or economic, who manifest gastric symptoms not unlike those of ulcer This is true not only of dispensary cases, but in hospital and private practice I know

of no scheme or system to enlighten you as to how to elicit these important facts from the patient The psychiatrist or psychoanalyst is often of great value The social service worker will certainly be of great assistance However, of greatest importance is the physician himself The family physician as well as the specialist should be able to "feel the family pulse" The physician who can become the family confidant or confessor will certainly affect the "cure" of many mythical ulcers *Gastric neuroses*, like many other neuroses, are social maladjustments The remedy may often lie beyond the powers of the physician, but the co-operation of the patient is readily obtained once the origin of the trouble is explained Social agencies and their workers are often used to great advantage

Simple Habitual Constipation—I call your attention to this condition because of its frequency and the relative ease of recognition Yet we often see many cases of constipation which are diagnosed as peptic ulcer Constipation often occurs in duodenal ulcer It is said by some to be an etiologic factor It may also result from the dietetic treatment of ulcer and finally it may appear as part of the picture of chronic colitis Examination of the history of the habits of the patient and the number and character of the stools will serve to make the diagnosis of constipation

Colitis—This applies to those patients who present gastric discomfort associated with attacks of abdominal pain, followed by diarrhea The diarrhea is trigger-like in character It comes almost immediately after food intake Subsequent to these attacks patients often develop periods of constipation at which time the periodic heart-burn after meals occurs Coarse foods—coarse vegetables, cabbage, celery, lettuce, spices, pickles—precipitate these attacks At times these patients may be cathartic addicts and present a story of alternating periods of gastric discomfort associated with cathartic abuse In many of these cases it is impossible to differentiate from the group which I have described to you as the "neurogenic chronic appendicitis" Atropin sulphate in moderate doses, followed by

a smooth diet, as recommended by Alvarez, is often very efficacious

With all these possibilities in mind and as far as possible eliminated by clinical and laboratory methods, I think one can safely assume that a diagnosis of peptic ulcer is most likely in the presence of definite periodic epigastric discomfort, associated with food intake and relieved by alkalis Localized tenderness, hyperacidity, blood in the stomach contents and stools, complete the picture. The absence of roentgenologic findings does not warrant the dismissal of the diagnosis of ulcer Such patients are best placed on ulcer management Repeated gastric and stool analysis should confirm or disprove the findings Roentgenograms repeated at six month periods and the patient should be under observation for a period of two years These are the type of patients which are usually dismissed only to appear later as cases of chronic duodenal ulcer

CLINIC OF DR EUGENE F TRAUT

RUSH MEDICAL COLLEGE

GLÉNARD'S DISEASE (VISCEROPTOSIS, ASTHENIC HABITUS) TREATED WITH INSULIN

For your consideration we have an unmarried woman thirty years old with generalized visceroptosis or Glénard's disease She comes because of extreme thinness and lassitude amounting to disability She had been forewoman in a bookbindery until the last year She has dragging sensations in the abdomen and a sense of stuffiness after eating

She has never known what it is to be well As long as she can remember she had been weak, and has suffered from an "upset stomach" She never gets enough rest She sleeps soundly and long, but is tired on arising She is never hungry She has rarely experienced appetite The thought of breakfast fairly nauseates her She has not eaten breakfast for years Lunch time or the dinner hour may find her with less aversion to food A few mouthfuls satisfy that Even her very small meals make her feel "stuffy" and uncomfortable She also complains of belching and flatus She does not use laxatives Occasionally diarrhea troubles her She sleeps nine or ten hours every night. She usually arises about 9 in the morning and has a cup of coffee At noon she has coffee and a sandwich At night she eats a little meat and vegetables She stays in the house except for very short walks She does a few light tasks about the house

She is 5 feet, 8 inches tall Six years ago she had reached the maximum weight of 99 pounds She now weighs 95 pounds in street clothes. Attempts at forced feeding have caused nausea and vomiting lasting for days Slight fever has been

noted at such times She is often weak and dizzy She faints She denies being "nervous" Headaches or spots before the eyes were never noted Colds are an uncommon experience with her They are never bronchial in type Cough, night-sweats, and hemoptysis are all denied Her throat is never sore Palpitation occurs on slight exertion

Menstruation did not start until fifteen years The periods have always been irregular She menstruates every second, third, or fourth month There are some cramps on the first day

There had been no feeding troubles in infancy She left school upon graduating from eighth grade at fifteen years

Of course she has been repeatedly examined for signs of tuberculosis Tuberculosis has been the punishment promised her if she would not eat more Four years ago she was taken to a hospital for an appendectomy She was having an attack of "upset stomach" She was sent home without any attempt at surgical treatment She was advised at one time to have her "kidneys suspended"

Her parents are of moderate stature and are thin The three other children in the family are of normal proportions None of her relatives or her associates have had tuberculosis

Here is a tall, thin young female, not apparently ill She is a sallow brunette In spite of her spare figure, she is distinctly not cachectic Her pulse is 92, her temperature is 99° F Her systolic blood-pressure is 100, diastolic 70

The eyes are deeply set, but not sunken The tongue is lightly coated and dry No abnormal pigmentation is seen in the mouth or on the lips The tonsils are small and smooth Three teeth are capped The thyroid is small and easily palpable in her long, thin neck

Her chest is long and narrow, a type called the paralytic or phthisical thorax The breasts are small and flat The scapula are "winged" Clavicles, ribs, and all other skeletal parts are prominent Corresponding with the thoracic contour the intracostal angle is acute

The heart borders are well within normal limits There are no murmurs or other abnormalities of the heart sounds

Expansion of the lungs is symmetrical and is of normal amplitude There are no abnormal areas of dulness, no adventitious sounds are heard on repeated examination There is nowhere any prolongation of either respiratory phase

The liver extends 2 cm below the ribs on deep inspiration The spleen is easily palpable It does not seem to be enlarged Both kidneys descend very low on inspiration The hand can be inserted above the upper pole of the right kidney and the organ held down through expiration The whole colon is palpable and slightly tender The cecum feels "juicy"

The hands are long and thin You see the bony structures prominently in both upper and lower extremities The fingers do not tremble Myotonic contractions of the arm muscles cannot be induced by squeezing them

The uterus is rather small and mobile It has normal proportions The ovaries are not felt

The urine is clear Its specific gravity ranges from 1 004 to 1 020 Neither albuminuria nor glycosuria has ever been detected

The hemoglobin is 70 per cent She has 3,680,000 erythrocytes They have the normal configuration and color There are 11,000 leukocytes The differential count indicates nothing abnormal in the proportions of the various white cells

Her stomach was empty thirty five minutes after eating a piece of toast and 400 c c of water Aspirating twenty minutes after such a meal returned 40 c c of stomach contents There was no free acid Repetitions of the Ewald breakfast have always shown an absence of free hydrochloric acid Aspiration of a "motor meal" at the end of seven hours gave no residue

The Wassermann test on the blood is negative Her basal metabolic rate was + 4 per cent According to *x* ray films, four of her teeth had filled roots

An *x* ray study of the chest showed no lung pathology The heart is "dropped" or that type frequently described in "neurocirculatory asthenia." It hangs more perpendicularly than is usual Under the fluoroscope the stomach seemed to be markedly ptotic The lesser curvature was below the umbilicus

Six Months After First Observation —After her first presentation in this clinic Miss D was sent home with written instructions for an unweighed diet affording about 3000 calories This included three meals and milk and cream between meals and on retiring For the last five months whole ovary has been injected intramuscularly twice weekly Menstruation now occurs every twenty-eight days She has taken 30 drops of dilute hydrochloric acid before, during, and after meal time

Two months of such a régime failed to increase the weight The fatigue was not relieved She was unable to eat the large amounts of food without nausea or even vomiting She meas-

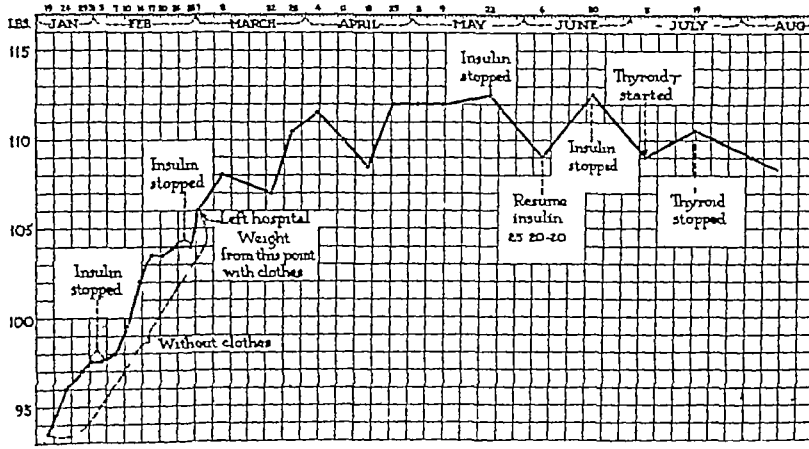

Fig 78 —Chart of weight changes and periods of medication Insulin administered from January 19th to May 9th except for two four-day periods as charted

ured her temperature three times daily It did not rise above the normal readings Three months ago she was admitted to the hospital, practically in the same condition as when first seen Without clothes she weighed 93 pounds Her blood-pressure was 112/68 Her achlorhydria continued Her stools were always formed They reacted negatively to benzidin test for blood The urine showed no abnormalities In 10 c c of fasting blood there were 10 mg of urea nitrogen, 3 4 mg of uric acid, 1 3 mg of creatinin, 30 4 mg of non-protein nitrogen, 104 mg of glucose, and 470 mg of sodium chlorid Her carbon

dioxid tension was 63 6 volumes per cent Her daily temperature in the hospital varied from 97° to 98 6° F Her pulse varied from 80 to 104 on the same date

For the first three weeks in the hospital the program pursued before entrance was continued Then 5 units of insulin were injected two hours before each of the three meals Her visiting acquaintances noted her eating more than ever before She gained 2 pounds each week We ascertained that upon an injection of 5 units of insulin at 10 A M her blood sugar would fall from 108 to 97 mg at lunch time two hours later After two weeks injections of sterile water were substituted for the insulin without the patient's knowledge Appetite immediately disappeared

The insulin injections were resumed, the dose being increased to 10 units Appetite increased with the larger doses She had several slight "reactions" before her meals, leading us to shorten the intervals between injections and mealtime to one hour She had by this time learned to take her own insulin and her injections of ovary

Breakfast still remained the most difficult meal To increase the appetite for breakfast the morning dose of insulin was increased to 15 units This lowered the blood sugar level from 107 mg (6 30 A M) to 46 2 mg (7 30 A M)

Discontinuance of the injection of whole ovary was followed by complete amenorrhea lasting two months Injections of ovary were then resumed and continued They have led to regular menstruation

Sterile water was again substituted for insulin The decrease in the amount eaten shows itself in the weight table On resuming insulin the doses were increased to 20 units before breakfast, 15 units before dinner, and 15 units before supper

As you see her today she is still thin, but has lost the haggardness of the first examination Her weight without clothes has increased during the hospital stay of forty-one days from 93½ to 106 pounds Her average pulse rate has decreased from 106 of the first weeks in the hospital to 76 The temperature

maintains a higher average She is stronger and more active We have prescribed a liberal amount of outdoor walking during her hospital stay A recent Ewald showed 10 points of free hydrochloric acid Acid by mouth was discontinued She is leaving the hospital today, instructed to eat the same large amounts of food served in the hospital and to inject insulin on a schedule of 20–15–15 She is also injecting whole ovary twice weekly

Ten Months After the First Appearance in Clinic Five Months After Dismissal from Hospital—In our efforts to increase her appetite the insulin was advanced to 30–20–20 Remember that she has no diabetes Starting at 7 A M she took the doses one hour before meals Her only "reaction" has been hunger This has been least before breakfast Her breakfast frequently affords only 20 grams of glucose because she will not eat more The hydrochloric acid has been resumed, as a recent Ewald again showed achlorhydria Since taking insulin there has been a quite steady gain in weight from a low point of $93\frac{1}{2}$ to a maximum of $112\frac{1}{2}$ pounds five months later Each interruption of the insulin therapy is marked by a plateau or a fall in the weight curve She refused last month to take any more insulin Whole thyroid, 3 grains, was given by mouth for one month As anticipated, its only effect was the production of a slight tremor and tachycardia

She is stronger and has returned to work

Insulin has been used extensively, especially in Europe, to increase the weight of marantic children According to the literature, under insulin thin insane patients have made substantial gains in weight Insulin is being used to combat the anorexia of tuberculosis

Its mode of action in fattening the non-diabetic is not quite clear There is no evidence that patients with visceroptosis lack the ability to make any amount of insulin required They do not show glycosuria nor a hyperglycemia on ingestion of large amounts of carbohydrates We propose that the mild hypoglycemia following injection of insulin provides an incentive to eat and gives an appetite

Our rationale was to engender an eating habit, to accustom the stomach to larger amounts of food, and thus to provide an excess of calorigenic material This excess would be stored as fat We have only partly succeeded Our patient has gained weight and strength. Her symptoms are ameliorated

CLINIC OF DR HOWARD WAKEFIELD

RUSH MEDICAL COLLEGE

MILDER TYPES OF CORONARY ACCIDENTS

IN 1912 Dr James B Herrick described very clearly the clinical features of sudden occlusion of the coronary arteries of the heart In 1918 he presented some of the electrocardiographic findings which are usually associated with these accidents

In his original paper Dr Herrick divided the cases of coronary accidents into four groups as far as outcome was concerned His classification at that time was as follows

1 Cases of instantaneous death

2 Cases of death within a few minutes or a few hours after the obstruction

3 Cases of severity, but where death is delayed for several hours, days or months, or where recovery occurs

4 A group that may be assumed to exist, embracing cases with mild symptoms, *i e*, a slight precordial pain, ordinarily not recognized, due to an obstruction in the smallest branches of the arteries

It is this fourth group of cases of Dr Herrick's which I am calling the milder or subacute types of coronary accidents, and about which I wish to say a few words and to present several typical patients

Clinical Features —The fundamental feature about the pathology of coronary accidents is the sudden plugging of one of the coronary arteries either by a thrombus or an embolus usually a thrombus If one of the coronary vessels or its larger branches is occluded, we get the well known symptoms of acute obstruction, and our patient, as far as outcome is concerned, falls into one of the first three groups presented earlier It is

natural to assume that if the smaller branches of the coronaries are obstructed the symptoms will be milder, in fact, some of the smallest twigs might be occluded and the accompanying symptoms so slight that the patient would not even consult a physician These subacute cases occur in patients, usually males, past the middle span of life Especially common is the presence of a vascular hypertension or the angina pectoris of effort These individuals until their coronary accident may give a history of apparent perfect health, but on examination there may be some evidence of generalized arteriosclerosis In many, however, the sclerosis may be purely a local process, involving only the coronary vessels These subacute or milder attacks may occur in patients who have suffered a major attack and survived, only to be reminded at a later date, by the symptoms of a subacute attack, that the disease is still present in their coronary vessels

The subacute attack is usually ushered in by a sudden attack of precordial pain Sometimes the pain may be substernal, or epigastric, or run transversely across the chest and even radiate into the neck or one or both arms If the patient has been a sufferer from angina pectoris, he may state himself that this attack of pain is different from anything like the pain he has suffered in the past He may locate the pain in a different part of the chest The pain, after it appears, usually lasts for a few hours, maybe a good part of a day, and in some cases for several days or more The usual relief which the angina patient receives from nitroglycerin does not come The attacks may come on while the patient is at rest, or when under emotional distress, or walking, but the three features which differentiate this coronary pain from the pain of angina of effort are the facts that its duration is much longer than the pain of angina pectoris, it is not relieved by nitroglycerin, and that in many cases the patient recognizes the new type of pain, especially as regards its location If the pain should come on while the patient is walking and relief did not come when he stopped and rested a while, this would certainly be a new experience for the old anginal sufferer and probably would be enough of a scare to make him consult his physician almost immediately

Nausea may be present, but vomiting is not a constant finding. As these sufferers from subacute attacks usually do not die, autopsies experienced on these cases is not available, but we may assume that occlusion of a fine coronary twig produces a very small infarct which usually never reaches the visceral pericardium, so that the pericardial friction rub which is a common feature of the acute cases is usually absent in the milder types. Neither do the evidences of a failing heart come on suddenly or even gradually as they do in the graver cases

A slight rise in the body temperature is usually present, about 1 or 2 degrees above normal A leukocytosis in the neighborhood of 10,000 to 15,000 is usually present In some cases the patient will become short of breath rather suddenly Some degree of chest pain is usually present, in a few cases dyspnea may be the only symptom, *i e*, the coronary accident is silent as far as pain is concerned The sudden appearance of dyspnea plus chest pain should always make one consider the possibility of a coronary accident, either acute or subacute Most of these subacute cases continue about their work and usually do not go to bed for their illness

The electrocardiogram is very valuable in these cases where a previous tracing has been taken before the onset of the subacute coronary accident. In the tracing taken after the accident we may see inverted T waves in leads I or II, or both The Q R S interval may be prolonged, the Q R S waves individually may be split. Any way, the important feature is the sudden appearance in the electrocardiogram of findings suggestive of myocardial change—findings which did not exist in previous tracings The presence of the Pardee wave practically establishes the diagnosis of a coronary accident. On account of the valuable information afforded by the electrocardiogram in coronary accidents the electrocardiographic method should be routine in cases of angina pectoris, hypertension, and cardiac disease in general, for we never know when a coronary attack may come on and complicate the picture presented by any of the cardiovascular diseases And, after all, subacute coronary attacks, like the acute affairs, are not rare diseases In fact, they are

more common than one would suppose But one must keep the possibility in mind if he is to diagnose it

I shall now show two patients who present the clinical features of the milder types of coronary accidents

Case I.—*The first patient is a white man, married, and sixty-four years* old He weighs 220 pounds and is the director of a large organization He had pneumonia in childhood and his appendix was removed some years ago, otherwise he has enjoyed good health all his life He has worked hard all his life His habits are good For the past year he has complained of epigastric pain on walking The pain is severe enough at times to make him stop walking and then he gets almost immediate relief The pain sometimes appears over the precordium and radiates into the neck and the left arm He is slightly short winded on climbing steps The pain is sure to come on if he walks immediately after a heavy meal He has had no acute attacks of pain which lasted for some time in which it was necessary to call in a physician for aid Nitroglycerin, 1/100 grain, gives him relief The pain seldom lasts any longer than *five minutes* His systolic blood-pressure is 200, the diastolic 110 His teeth are in poor condition, many are loose The heart is slightly enlarged to the left The heart tones over the aortic area are accentuated and there is a slight systolic apical murmur There is a postoperative hernia in the right lower abdominal quadrant The physical examination is otherwise negative The urine is normal In this patient it is perfectly evident that we are dealing with angina pectoris of effort and vascular hypertension

The electrocardiogram in his case shows a left heart preponderance, otherwise a normal tracing

For the past year things have been going very well with him As long as he walked slowly and rested after his meals he felt perfectly comfortable He considered himself a well man On the morning of October 25, 1928 he was in conference with some of his officers Some big business deals were being put over and he was rather excited, although comfortably seated in a chair All of a sudden he experienced a dull heavy pain under the lower end of the sternum The pain was not severe enough to make him call in a physician He stayed on and completed the business of the conference, but when he left the pain was still present He himself noticed the important fact that nitroglycerin did not relieve the pain this time as it usually had The pain lasted all night, but was somewhat milder the following morning In the conference room he felt slightly nauseated when the attack began, but did not vomit He was never short winded and no cough developed Several days later he reported at the office On the evening of his attack he said he felt feverish In the office his temperature was 99 5° F and the white count was 10,400 His systolic blood-pressure was 174, the diastolic 120 On all previous occasions during the past year his systolic pressure has been 200 or over The physical examination was otherwise negative The electrocardiogram at this time revealed a cone-shaped inverted T wave in lead I, which is a new finding in his case

To summarize then, in this man we have sudden substernal pain, heavy and dull in character, lasting for twenty-four hours or longer, not relieved by nitroglycerin, a slight fever, a leukocytosis, a fall in the systolic blood pressure, some nausea, and the sudden appearance of an inverted T wave in lead I of the electrocardiogram It is on these facts that I think a diagnosis of subacute coronary accident is justified The patient is not laid up, does not go to bed, but continues with his daily work There has been an occlusion of one of the finer twigs of one of the coronary arteries

Case II.—The second patient is a white man fifty six years old He weighs 185 pounds and his health has been very good all his life. He has had a periodic physical examination every six months for the past ten years His examining physician declared he was always in good condition his urine normal and his systolic blood pressure was usually about 140 On September 13 1928 he went to a baseball game feeling perfectly well. While being pushed about in a crowd at the ball park he suddenly had a severe pain in the jaw just in front of both ears He immediately became short of breath felt weak, and dizzy As he expressed it, he began to spit cotton There was absolutely no pain in the chest and he had never had a similar attack in his life before He sat through the baseball game but felt very uncomfortable the pain in the jaw continued He went home in an automobile did not take any supper and went to bed without calling a physician He was restless all night and got very little sleep

The next morning he felt very weak, was sweating all over and was still short winded The pain in the jaw still continued but was milder After eating a light breakfast he went to a hospital of his own accord and was put to bed. He was kept in bed for one week and told that he had a weak heart. He stated that while a patient in the hospital he examined the chart and found that his blood pressure had fallen to 106 systolic. His temperature was 99 3 F and the white cells were increased in number how much he did not know

Since his discharge from the hospital he is still short winded on climbing steps and the pain is liable to reappear in the jaw if he walks too fast On October 18 1928 the shortness of breath and pain in the jaw on walking were his two chief complaints At that time his blood pressure was 100/64 in the left arm and 100/62 in the right arm in the recumbent position The white count was 10 400 cells per cubic millimeter The urine was normal The electrocardiogram showed an inverted T wave in both leads II and III The QRS complex in lead III was of low voltage.

The fluoroscopic examination of the chest revealed a cardiac apex which was unusually pointed and strongly suggested the possibility of a parietal aneurysm From all of these facts it is rather conclusive that this patient had a subacute coronary accident on September 13 1928

To summarize then, the clinical features of subacute or milder types of coronary accidents are as follows The disease usually occurs in men past fifty years of age, men who give a history of angina pectoris, hypertension, or have evidence of arteriosclerosis The attack of pain comes on suddenly, lasts for hours, and is not relieved by nitroglycerin Many of these attacks come on while the patient is at rest There is a fall in the systolic blood-pressure, a slight rise in the body temperature, and a mild leukocytosis Electrocardiograms usually show evidence of myocardial injury, such as inverted T waves in either leads I or II, or both

After some time the fluoroscopic examination may add a bit of evidence such as a parietal aneurysm The features of collapse, shock, pericardial friction-rub, and visceral evidence of cardiac decompensation—features characteristic of the acute coronary accidents in which a much larger vessel is obstructed—are lacking

Patients usually do not die from the effects of the milder types of coronary accidents, so that postmortem evidence is not available, but nevertheless the condition is a real clinical entity and much more common than is suspected To make the diagnosis one must keep the condition in mind

I am indebted to Dr James B Herrick for the privilege of presenting these two patients whom he first saw in his private practice

CLINIC OF DR WALTER S PRIEST

MICHAEL REESE AND CHICAGO MEMORIAL HOSPITALS

HYPERTHYROIDISM ASSOCIATED WITH ABDOMINAL LESIONS

THESE 2 cases are presented not primarily to discuss hyperthyroidism, although the first patient was sent to the thyroid service to prepare for thyroidectomy, but rather as examples of those complex problems in medicine which confront us at times

Case I.—This patient a married woman of fifty was admitted to the medical service (Dr Solomon Strouse) November 20 1928 complaining of weakness and excessive sweating one year sharp pain in the right upper quadrant six to seven weeks ago associated with feeling of fulness and nausea after eating and loss of appetite The excessive sweating was present both day and night amounting at times to drenching night sweats Associated with the upper abdominal pain was a chilly sensation followed by fever, and occasional vomiting of yellow fluid containing food There was no jaundice. The severe pains lasted about three or four weeks but the feeling of fulness and nausea after eating persisted She lost about 20 pounds in weight. Her trouble was sufficiently severe to keep her in bed most of the time during the six or seven weeks prior to admission The most significant point in the past history was a panhysterectomy performed nine years ago seemingly for urinary incontinence, but later she was told she had a tumor of the uterus. Artificial menopause resulted The incontinence was relieved, but frequency of urination both day and night and burning persisted Four years ago she had severe pain and stiffness in the back.

Examination on admission showed a well nourished middle-aged woman lying quietly in bed and not appearing acutely ill Temperature 99.2 F pulse 108 respirations 24 Fetor oris coated tongue, enlarged and red tonsils were the significant findings in the mouth The thyroid, especially the isthmus was enlarged to palpation although the patient had not been aware of any enlargement of the neck. There were no pathologic eye signs noted but a fine tremor of the fingers and tongue was present The pupils reacted to light and accommodation and convergence was good The lungs were clear except for a few fine crackling rales at the left base posteriorly The heart outline was within normal limits rhythm regular The aortic second sound was slightly accentuated and there was a soft blowing systolic murmur at the

apex, not transmitted Blood-pressure was 146/70 Tenderness on pressure was elicited in both upper quadrants of the abdomen and slight resistance was present to the right of the epigastrium There were external and internal hemorrhoids

Urinary findings were normal The white-cell count was 11,400, otherwise the routine blood examination was normal Wassermann was negative She was unable to retain a gastric test-meal, but repeated examinations of vomitus after the test-meal showed an absence of free hydrochloric acid and a total acidity ranging from 43 to 0 One stool examination was positive for occult blood, but in view of the hemorrhoids this was of doubtful significance Roentgenologic examination of the gastro intestinal tract showed several large stones in the gall-bladder and a colitis

During the first five days of her stay in the hospital she had a low grade fever ranging from normal to 100 4° F , the pulse remained rapid, 110–120, in spite of bed rest, and she vomited almost daily and complained of abdominal distress On the sixth day the surgical consultant advised cholecystectomy, the diagnosis being chronic cholecystitis and cholelithiasis Certainly a rational and proper procedure in view of the findings However, the next day a basal metabolism reading of plus 61 was obtained Attention was then directed to the findings possible of interpretation in the light of this latest laboratory finding Going back we have weakness, sweating, fine tremor of the hands and tongue, a tachycardia which persists after bed rest, a palpably enlarged but not nodular thyroid, a soft precordial systolic murmur, a blood-pressure of 146/70, even a low-grade fever which is not uncommon in thyrotoxicosis If this patient had a thyrotoxicosis, a major abdominal operation would have been most unwise. Accordingly another metabolism test was made two days later This showed a rate of plus 67 Since both tests were satisfactory so far as the graph and co-operation of the patient were concerned, a diagnosis of thyrotoxicosis in addition to gall-bladder disease seemed justifiable Accordingly, on the advice of Dr Strouse, the patient was transferred to the thyroid group service for further study

The thing which attracted my attention first when I saw the patient was the pigmentation of the skin It was not the mottled reddish-brown pigmentation of thyrotoxicosis, but a dark uniform brown or tan, most marked in the collar area, the forearms, axilla, under the breasts, over the upper inner surfaces of the thighs, and in the gluteal folds The remainder of the skin had a *café au lait* color The patient was quite positive that this pigmentation was a recent thing, dating it to about the time of onset of the abdominal pain The scleræ were not icteric The liver was palpable in the epigastrium, but not tender In the gall-bladder area could be felt a small, hard, rough mass, slightly tender, which was thought to be the gall-bladder fundus containing a stone Both cecum and sigmoid were tender The patient was worried about herself and family and cried easily, but no more so than many hospital patients In addition to the findings noted while on the general medical service, palpitation and pounding of the heart, sensations of heat and cold, pressure in the neck, nervousness, irritability, apprehension, only fair ability to concentrate, poor memory, restlessness, apathy, muscle weakness, lack of emotional control were elicited as further evidence of thyro-

toxicosis However, with the exception of the restlessness fatigability muscle weakness and emotional control these symptoms were not marked

The abdominal pain fever and vomiting persisted Blood-chemistry on December 14th showed non protein nitrogen 40 urea 1 sugar 133 The direct van den Bergh was negative the indirect faintly delayed positive She was given Lugol's solution 15 minims three times daily beginning December 6th and continuing to December 27th On December 12th the basal rate was +58.3 The temperature at the time of this reading was 100 4 F rectally Deducting 7 per cent for approximately 1 degree fever the actual basal reading would be +51 The temperature at the time of the two previous readings was normal The pulse rate was not affected continuing to range from 96 to 122 throughout the time Lugol's solution was given and it did not seem to me that her nervousness or emotional instability were improved Certainly the weakness was not but this was explained by later developments.

On December 20th slight pitting of the abdomen chest arms and legs was noticed On December 26th she became suddenly worse complained of extreme weakness was drowsy almost stuporous The pulse was weak and rapid Pupils reacted sluggishly to light and were dilated The tongue was red dry and coated The pigmentation of the skin was deeper She had evidently lost considerable weight Lungs were clear the heart tones weak but no enlargement could be made out The blood pressure had dropped to 84/60 The abdomen was soft flat not tender Urine was scant contained a trace of albumin and the phthalein output was only 6 per cent. in two hours. Rectal temperature was 98 6 F x Ray of the chest on December 28th showed the right half of the diaphragm immobile and held very high Non protein nitrogen was 80 creatinin 2 uric acid 10 sugar 133 The possibility of adrenal pathology was considered as some cases of Addison's disease associated with hyperthyroidism have been reported but on account of the high immobile diaphragm the possibility of a subphrenic abscess was thought to be sufficiently good to warrant exploration Accordingly laparotomy was performed at 5 P M by Dr D C Straus

The liver was found to be studded with hard nodules and there were nodules throughout the abdomen The gall bladder contained two large stones No abscess was found The abdomen was immediately closed the diagnosis being diffuse carcinomatosis She died December 31st

The organs removed at autopsy are now presented for your inspection The liver presents the most interesting picture You will note the large tumor mass occupying most of the right lobe also the smaller masses scattered throughout the liver These masses are compact homogeneous grayish white in color and not definitely outlined from the adjacent liver tissue. The large mass was firmly adherent to the diaphragm accounting for the x ray finding of an immobile diaphragm Next you will note the large masses of tumor tissue along the mesenteric attachment of the intestines Tumor masses (lymph node metastases) were also found along the common and hepatic bile-ducts, and retroperitoneally along the aorta. The pelvic parietal peritoneum was thickened and studded with nodules The gall bladder contained the two large stones you see here which were felt before operation The spleen you see contains several tumor nodules The head of the pancreas

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feels firm and these lymph-nodes which were adjacent to it contain tumor tissue The kidneys show some clouding and swelling of the columns of Bertini, but do not grossly account for the suppression of renal function The adrenals are small, the medulla appears cystic, and the entire organ rather atrophied This may in a measure account for the pigmentation of the skin The thyroid weighs 40 grams, there are no nodules, the cut section has a diffuse amber colloid appearance, and the organ appears to be made up of numerous colloid droplets The heart and aorta are especially interesting in view of the physical findings You will note the marked degree of intimitis of the proximal portion of the ascending aorta In places the intima is calcified, sclerosed, and even ulcerated These changes extend down to the valves and surround the mouths of the coronary vessels The valve leaflets themselves are not involved in the process, however When you recall the definite and constant murmurs heard in patients with syphilitic and sclerotic aortitis with far less gross pathology it is difficult to understand the vague and certainly inconstant murmur in this case Furthermore, the only time a murmur was heard, it was at the apex and not transmitted The mitral valve, as you see, is somewhat atheromatous

The condition appears to be a primary carcinoma of the liver but we must bear in mind the pelvic tumor which was removed four years ago

Case II.—This second patient, also a woman, was referred by Dr G J Hagens She was sixty years old and had been troubled with abdominal discomfort, inability to eat solid food, loss in weight and strength for about a year The first day I saw her (August 4, 1928) she had had a profuse epistaxis which required packing to control Examination at that time (in the home) showed a rapid but strong pulse, blood-pressure 172/80, temperature 100 2° F, marked cachexia, a palpable, enlarged, nodular thyroid, slight enlargement of the heart to the left, a blowing systolic murmur at the apex, transmitted toward the base, *i e*, heard with about equal intensity over the precordium to the left of the sternum, scattered moist râles throughout the right lung posteriorly, a moderately distended abdomen, slight edema of the ankles Chief interest centered in the abdomen In the outer portion of the right upper and middle quadrants was a hard irregular nodular mass the size of an average grapefruit It was not attached to the anterior abdominal wall nor did it seem to be continuous with the anterior surface of the liver, the edge of which could be felt about a fingerbreadth below the costal margin It moved with respiration and was quite tender It did not fluctuate

After considerable persuasion the patient finally consented to enter the hospital for detailed study and was admitted to the Chicago Memorial Hospital on August 13th

In addition to the points mentioned above the detailed history brought out the following facts A progressive loss of 50 pounds in the past year, anorexia of over four months' duration, a known hypertension of about eighteen months, intermittent attacks of lower abdominal pain of seven to eight months' duration, becoming steady two weeks prior to admission, sharp and sometimes associated with nausea, but never vomiting, constipation of many years' duration, occasional "black" stool, a dry cough of three

to four months duration gradually becoming worse sense of fulness in the throat (thyroid region), and a very dry throat and tongue. About three weeks before admission she had a chill lasting about two and one half hours followed apparently by fever Except for an attack of jaundice twenty five years ago the past history was unimportant. The teeth had all been extracted in March, 1928 The admission examination showed pulse 100 respirations 20 temperature 100.2 F blood pressure 146/70 The apex impulse was diffuse heaving forceful There was slight collar pigmentation also some of the forearms a fine tremor of the hands and tongue and marked muscle weakness. Eye signs were absent Otherwise the findings were the same as above

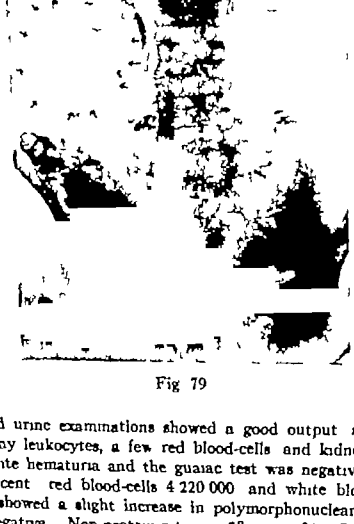

Fig 79

Repeated urine examinations showed a good output a faint trace of albumin many leukocytes, a few red blood-cells and kidney cells There was no definite hematuria and the guaiac test was negative. Hemoglobin was 70 per cent red blood-cells 4 220 000 and white blood-cells 14 000 Differential showed a slight increase in polymorphonuclear cells Wassermann was negative Non protein nitrogen 38 sugar 91 Stools contained a trace of blood (chemical) Sputum was negative for tubercle bacilli Basal metabolism rate was 53 per cent on repeated examination At the time of the first test the temperature was 99 F at the time of the second 98 6 F During her stay in the hospital there was a daily afternoon rise in temperature, the highest being 101 4 F Allowing 7 per cent for each degree of fever this could account for not more than 21 per cent of the metabolic rate

increase at the most, and this is probably a liberal figure, since at the time the tests were actually run the temperature was normal or practically so and did not rise for some hours afterward The chief interest centered in the gastro-intestinal x-ray examination Figure 79 is a plate made six hours after taking the barium meal Although swallowing the barium mixture had not induced coughing, the possibility of an esophageal fistula was considered Accordingly, a second meal was given and its progress carefully observed fluoroscopically The esophagus was clear, the stomach contour normal Shortly after entering the duodenum a column of barium was seen to ascend at right angles to the cap for about 10 cm and stop Shortly afterward the branch-like arrangement seen in Fig 79 appeared Plates were taken immediately, one of which is reproduced here (Fig 80) The outlines of the abdominal mass can

Fig 80

be seen (arrows) Some of the barium appears to be within the mass, some of it within the liver shadow Dr Landau mentioned that he had seen a similar condition in a patient at the Oak Forest Infirmary The patient refused to permit an exploratory laparotomy and remained in the hospital only six days The pulse-rate ranged from 96 to 100, corresponding to the slight elevation in temperature Those of you who were at the clinical conference when this patient was first presented will remember that opinion as to the origin of the mass was divided between a neoplasm, probably of the gall-bladder with obstruction of the cystic duct, and an enormously dilated common duct which permitted of regurgitation of the duodenal contents, and

an old cystic duct obstruction with subsequent hydrops of the gall bladder which became adherent to and finally ruptured into the duodenum in the meantime becoming plastered over with omentum and adhesions until the mass, as we found it was formed I think it is impossible to say which is correct, as the symptoms could well be explained by either condition However we can say definitely I believe that the chill persistent low grade fever and the distress after taking solid food were caused by infection in the bile passages resulting from the accumulation of duodenal contents

At home the patient went steadily down hill losing progressively in weight, strength and mentality She developed a rectal fistula and on December 4th a parotid abscess This latter was due no doubt to the difficulty in keeping the mouth clean She was readmitted on December 6th and the abscess drained by Dr P S Clark For a time she seemed to improve, but finally became comatose and died December 16th Autopsy was refused

Discussion —In reviewing these 2 cases one question which interested me was whether we were dealing with hyperthyroidism plus an abdominal lesion or whether the symptoms and physical findings indicative of hyperthyroidism could be accounted for by the abdominal pathology In both cases we were dealing with low grade infections, and in one, and probably in both, with widespread cancer Here we have the weakness and loss in weight, fatigability, sweating and tachycardia accounted for The emotional instability and nervousness were not marked in the first case and in the second the mental deterioration obscured the picture. Tremor was not marked in either case and might be accounted for by the weakness Neither patient had an excessive appetite, but this symptom is of no value in considering hyperthyroidism because of the abdominal pathology The blood pressures while suggestive are not conclusive. In Case I the readings are not abnormal for the age and in Case II there was enough arteriosclerosis to justify a hypertension That brings us to the basal metabolic readings and the question which to me is unanswered Could the readings have been accounted for by the low grade infectious processes, or the extensive carcinomatosis, or a combination of both? It is probable that they could not be accounted for on the basis of infection alone On the other possibility we are attempting to throw some light by making metabolic tests on patients with carcinomatosis with and without infection The microscopic

sections of the thyroid of Case I will not help much, since she had had Lugol's solution, and it has been shown that under the influence of such medication the gland, although hyperplastic to begin with, reverts to normal microscopically Neither case had more enlargement of the gland than is frequently seen in this region in normal individuals One suggestive fact is the comparatively little effect of lugolization on the metabolic rate and other symptoms At least the effect was less than we are accustomed to observe in similar uncomplicated cases here

Assuming that the first case had not had cancer, what should have been the procedure? I think in view of our present knowledge the plan we were following was the correct one That is, to give the biliary tract infection a chancé to subside and at the same time prepare the patient for thyroidectomy, do the thyroidectomy first, then later, when the patient's condition warrants it, the cholecystectomy This plan, of course, accepts the coexistence of the two diseases which at present we would have to do, however much we might speculate on the possibility of a single explanation for the whole picture In the second case, knowing we were dealing with an abdominal mass, possibly cancerous, my plan was to lugolize the patient as though preparing for thyroidectomy, but at the optimum point to do the laparotomy If the impression of cancer were confirmed, then nothing further could have been done If the mass were not cancerous, then the indicated surgery would have been performed and we would have prepared to fight a postoperative increase in the thyrotoxicosis and do the thyroidectomy as soon after the first operation as conditions warranted [1]

[1] Microscopic sections of the pancreas from Case I showed a primary carcinoma of the head It is interesting that a primary lesion so small as to escape gross detection should have caused such extensive metastases

CLINIC OF DR M H STREICHER

RESEARCH AND EDUCATIONAL HOSPITAL OF THE UNIVERSITY OF ILLINOIS AND THE GRANT HOSPITAL

STRICTURE OF THE ESOPHAGUS

ESOPHAGEAL pathology has always been of extreme interest to me because of the associated anatomic considerations that so very frequently present themselves as explanatory factors I wish to present a case at this time which I believe is instructive in many phases

A white male chauffeur twenty-eight years of age weighing 138 pounds was referred to our clinic at the Research and Educational Hospital of the University of Illinois by the Board of Paroles of the State on January 4 1927 presenting the following symptoms Difficulty in swallowing of four months duration and inability to elevate the left arm above the shoulder level The patient states that on August 21 1926 while engaged in a friendly skirmish in the state penitentiary one of the inmates slashed the left side of his neck with a sharpened nail file considerable bleeding followed but the wound soon healed Ever since the accident he noticed the impaired movement of the left shoulder and difficulty in swallowing

Past History—Essentially negative.

Family History—Is of social interest inasmuch as it consisted almost entirely of pardons and jail sentences

Physical Examination—The patient was apparently well preserved and evidently enjoying life The lungs showed no findings and the abdomen was entirely negative

Essential Findings—There was a deep bluish jagged scar extending horizontally from the left posterior border of the left sternomastoid muscle to the right border of the laryngeal cartilage The larynx was markedly deformed and the skin presented several areas of anesthesia. The upper portion of the sternocleidomastoid muscle was definitely retracted proximally thus sharply separating its lower segment The trapezius muscle showed marked signs of atrophy The left shoulder was extremely lower than the right

Urinalysis—Negative

Blood-count—Showed a mild secondary anemia apparently due to insti tutional diet rather than to loss of appetite The Wassermann reaction was

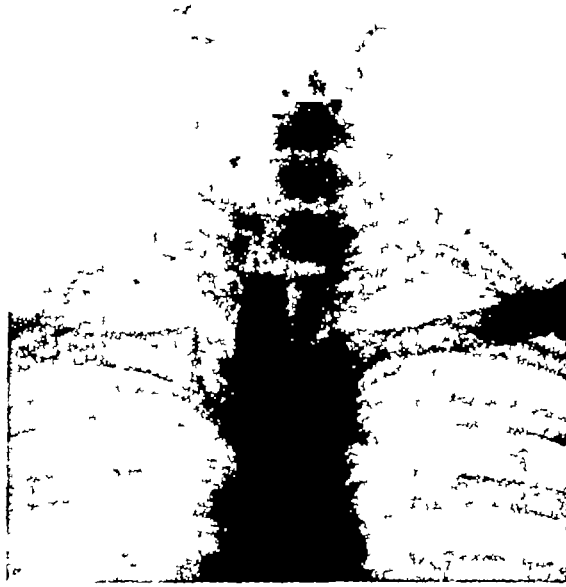

Fig 81 —Esophageal stricture Shows the anteroposterior view of the esophageal obstruction at the upper portion of the esophagus

Fig 82 —Esophageal stricture Shows the lateral view of the esophageal obstruction

negative. A roentgenologic examination made on January 18 1927 showed an obstruction above the epiglottis.

Esophagoscopy—Shows fixation of the left vocal cord and a definite constriction on the left side in the first portion of the esophagus at the cricopharyngeal curvature. The cicatricial stricture was markedly elevated and pale in color At the same sitting a 20 F bougie was passed with considerable difficulty and subsequent dilatations were attempted. Frequent dilatations were made—the largest bougie passed was a 27 F

Discussion—The most frequent cause of cicatricial stenosis of the esophagus is accidental swallowing of caustic alkali The cicatricial stenosis of the esophagus from instrumentation comprises a very small percentage of all strictures of the esophagus. The symptoms are those of progressive dysphagia, loss of weight, and regurgitation The regurgitation may set in at short intervals after food intake, depending upon the site of the stricture, the dysphagia becomes evident first upon intake of dry food or solid food, and as the disease progresses and the stenosis becomes more marked difficulty in swallowing is also initiated by fluids

The prognosis in these cases is discouraging The methods of dilatation are dangerous inasmuch as the pressure necessary to dilate a stricture almost invariably results in perforation of the esophageal wall With the aid of the esophagoscope bougienage treatments are favorable, depending on the degree of fibrous scar formation and the site of the stricture In tightly stenosed cases if a gastrostomy is done early good results may be expected, the advantage being that the esophagus is put at rest and the accumulation of scar formation thereby arrested

In this case the inability to raise the left arm above shoulder level, the retraction of the sternocleidomastoid muscle, and atrophy of the trapezius would naturally imply injury to the third and fourth cervical nerves and the spinal accessory respectively The patient was observed at the clinic for about four months, during which time he was dilated about once weekly, the largest bougie passed being 22 F He lost considerable weight and became weak A gastrostomy was advised as a prophylactic measure, but the patient did not wish to accept the advice, and did not return for further observation

A SILENT CARCINOMA OF THE STOMACH

R. H a white male miner fifty years of age, came to our clinic on October 22 1928 stating that a neighborhood physician had been treating him for constipation for fifteen years, but that he became discouraged in the past two years inasmuch as his treatments did not help him any more His essential complaints were epigastric distress varying from one to three hours after meals with considerable exaggeration of the distress by *fatty foods*.

Fig 83 —Carcinoma of the stomach. Shows a large number of filling defects along the lesser curvature of the stomach. The arrow points to the lesser curvature of the stomach

He also complained of abdominal distention dyspnea on exertion nocturia, and swelling of the lower extremities In the last two years he had lost about 60 pounds The past history is essentially negative.

Physical Examination —On examination patient appeared considerably older not acutely ill and had a marked yellowish tinge in the skin, showing evidence of marked wasting The heart and chest were negative The

abdomen was flat and on palpation a large mass was felt in the left upper quadrant, extending to the level of the umbilicus, the mass was irregular in outline, hard, and tender on manipulation

Urinalysis —Negative

Blood Analysis —The red blood-cells number 2,640,000 white blood-cells 10,800, hemoglobin 45 per cent The Wassermann reaction negative

A roentgenologic examination made on October 27, 1928 showed that there were numerous irregular filling defects, finger print in character, ex-

Fig 84 —Carcinoma of the stomach Shows the same series of films six hours after intake of barium meal

tending into the lumen of the stomach, suggestive of malignancy of the stomach A gall bladder visualization outlined the gall-bladder very poorly

Stools were positive for occult blood in all specimens collected for nine consecutive days

Gastric analysis showed no free hydrochloric acid The total acidity was 10 degrees, the test for lactic acid was positive The contents were positive for blood, no Oppler-Boas bacilli found

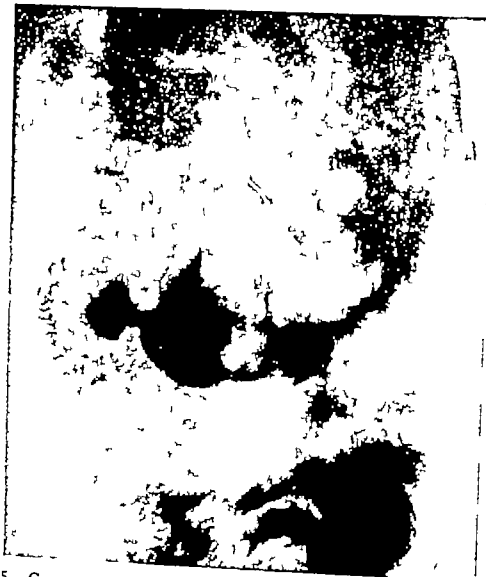

Fig. 85.—Carcinoma of the stomach. Showing the same series of films twenty four hours after initial barium meal

Discussion.—The diagnosis, of course, is obviously a carcinoma of the stomach. The most striking point deserving mention is the comparative negativity of symptoms in the presence of such an enormous mass, which involves practically the entire bed of the stomach save a narrow strip on the greater curvature. The absence of symptoms of obstruction is strongly suggestive that the malignancy in this case is either in the lesser or greater curvature and not near the pylorus.

About 55 per cent. of all gastric carcinomas involve the pylorus, 25 per cent. effect the lesser curvature and only 3 to 5 per cent. involve the greater curvature. Cancers of the lesser curvature or of the wall of the stomach may approach an enormous size with comparatively little or no interference

of proper emptying or gastric function in general About 80 per cent of all gastric carcinomas complain of loss of appetite The pain of cancer of the pylorus is invariably in the epigastrium, while that of malignancy of the lesser curvature is very frequently in the interscapular region Regurgitation or vomiting occurs in all cancers of the stomach as the disease progresses, but is a late manifestation in those of the lesser curvature or in carcinoma of the gastric wall It is important to look for early fixation of the tumor due to the fact that the lesser curvature has a tendency to early involvement of adjacent organs

Surgical intervention was not advised in this case, inasmuch as so little of the normal tissue remained with which to make an anastomosis

The patient was discharged from the hospital and advised to return for observation

ULCERATIVE COLITIS

N E., a white male, chauffeur twenty-eight years old, admitted to our clinic on November 22 1928 presenting the following essential complaints diarrhea of two years duration the number of stools varying from five to fifteen daily, containing visible blood mucus and pus he had considerable abdominal cramping especially marked at the initiation of a defecation reflex. In the past seven weeks he has lost about 25 pounds has grown progressively weaker, and was obliged to give up his work and be confined to bed. His temperature varied from 96° to 103 F and pulse from 72 to 124 Respiration 22 to 36

Physical examination is negative with the possible exception of a few suggestive râles in the left lung A roentgenologic examination of the chest showed some calcified nodules in the lower lobe with exaggeration of linear markings of the lower lobes.

Urinalysis—Negative

Wassermann reaction was negative.

Blood-count showed a moderate secondary anemia with a leukocyte count of 16 400 apparently explanatory of the toxic temperature.

Stools—A careful search for parasites was made but none was found

Proctoscopy examination revealed two terminal stages of ulcerative colitis so well described by Buie of the Mayo Clinic. The entire colon in the distal 15 inches was riddled with miliary abscess formation and ulcerations which bled readily upon the slightest manipulation. The mucosa of the colon in which no ulcers were present was markedly edematous and devoid of the natural pinkish velvety luster

Discussion—A smear was obtained through the proctoscope and cultured and subcultured bacteriologically The following bacteria were isolated from the culture Bacillus coli, B welchii, and a Gram positive diplococcus lancet shaped, belonging to the Streptococcus viridans group, this non hemolytic streptococcus bacterium gives negative reaction on lactose, maltose, mannite, salicin, and inulin.

A polyvalent autogenous vaccine was then prepared and given to the patient subcutaneously in progressively increasing doses at regular intervals. This treatment, however, did not suffice. The patient was placed on a low residue diet of high caloric content. Antiseptic irrigations of silver nitrate were begun These measures were combined with opium and tannic

acid medication by mouth in order to curtail the diarrhea and bleeding The patient rallied along, but grew worse, became more toxic, the temperature rose to 104° F , and vomiting ensued, so that he was hardly able to retain any nourishment

As a last resort a Mikulicz operation was performed and irrigations subsequently instituted through the cecostomy opening The patient got along very nicely for seven days, gaining weight, and remaining free of temperature One morning at six the nurse reported him dead in bed Unfortunately no autopsy was obtained, so we are still at a loss as to the definite cause of his sudden death

STRUCTURE OF THE THYROID GLAND IN TOXIC GOITER

RECENTLY the structure of thyroid in toxic goiter has come into the limelight again, with particular relation to the so-called toxic adenoma of the thyroid, and the separation of the thyroid-gland syndrome into two divisions (1) Exophthalmic goiter and (2) toxic adenoma (Plummer Mayo Clinic)

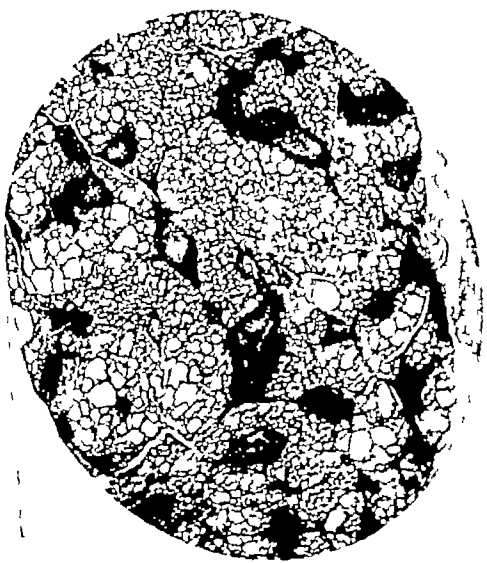

Fig 86

Reinhof and Dean Lewis, in material from Johns Hopkins Hospital, have from a pathologic viewpoint thrown definite doubt upon the validity of the toxic adenoma as a clinical entity

It has been shown by Reinhof that the structural form of the thyroid undergoes modifications which should reflect the changes of functional activity, indicated seemingly by the clinical im-

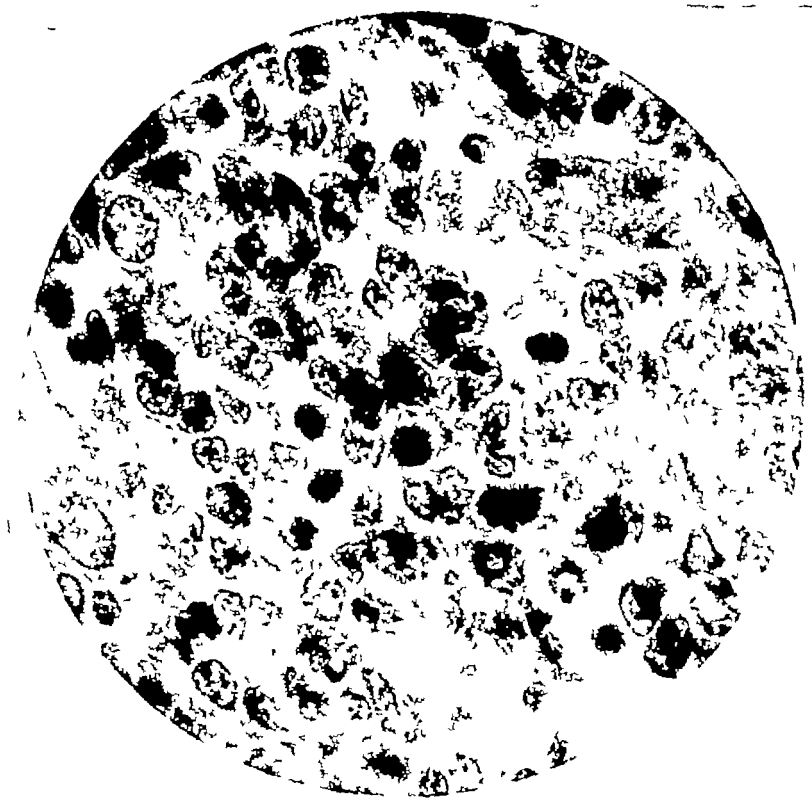

Fig 87

provement of the toxic thyroid under iodine therapy This structural modification seems to them to consist in the conversion of hyperplastic and hypertrophic gland into a simpler colloid type

One feature not usually accentuated in the consideration of the thyroid gland from a pathologic point of view is the lymphatic

hyperplasia that exists in the thyroid in some cases of toxic thyroid

The photomicrographs here shown are from cases of toxic goiter operated on my service at the Illinois Central Hospital, Chicago

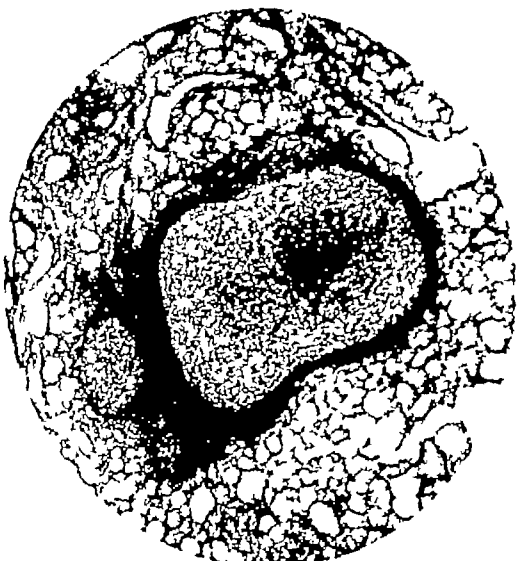

Fig 88.—A well-defined germinal follicle with invasion of the lymphocytes into the neighboring acini and between them The arrangement of cells suggests the presence within this germinal area of small acini The margins of the germinal area are well defined and the cells are seen in columns and deeply stained

The degree to which the lymphatic hyperplasia is carried, in some instances, is shown by the photomicrograph below

It is here seen that lymphatic follicles occupy a predominant part of the field The gland itself is of alveolar type, the acini being poorly filled with colloid There is little evidence of

hyperplasia or hypertrophy except on the part of the lymphatic elements

The central areas of these lymphatic germinal areas are made up of lymphoblasts, many actively mitotic figures are present

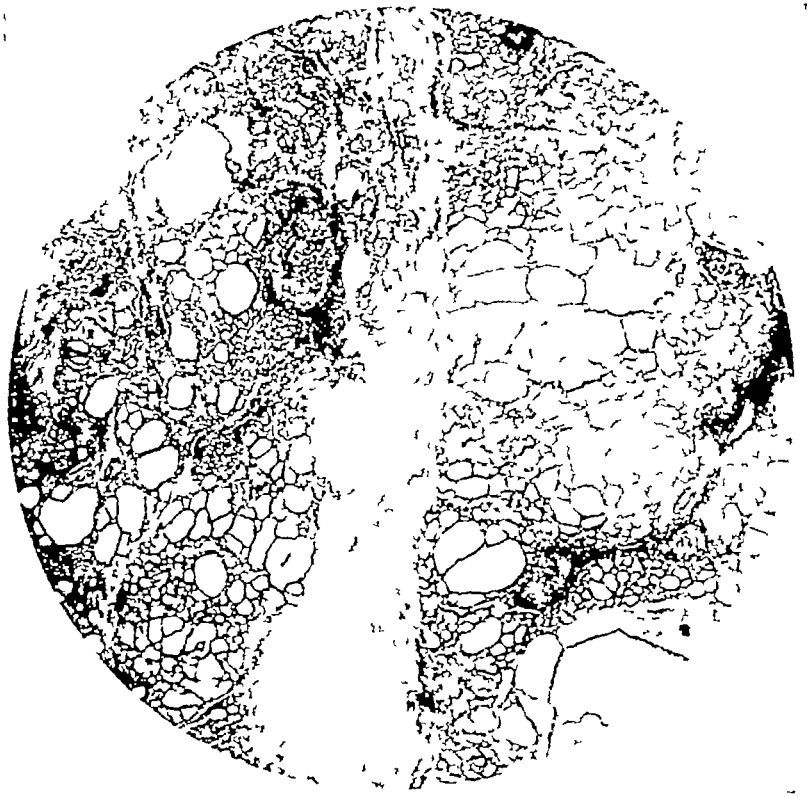

Fig 89.—Shows a thyroid of alveolar structure the acini lined with flattened epithelium and containing little colloid with areas of lymphatic hyperplasia and degenerative changes in the interstitial tissue

It is of interest that the lymphocyte count in some of the more toxic cases was occasionally very high (50 to 70 per cent small lymphocytes)

In 2 cases massage of the thyroid gland for ten minutes was followed by an increase of lymphocytes in the circulating blood In 1 case this increase was from 40 per cent prior to massage to

78 per cent six hours afterward. In [illegible] massage was attempted, the increase was [illegible] total. No control by massage elsewhere [illegible].

In 1 case of toxic goiter an increase [illegible] followed a fifteen minute massage [illegible].

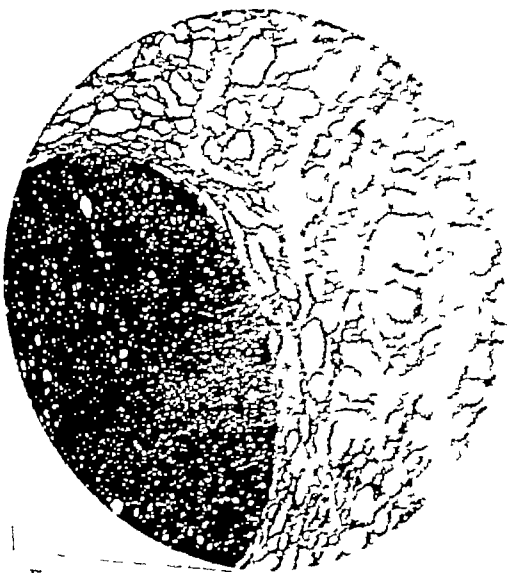

Fig. 90.—An area composed of closely packed [illegible] acini containing no colloid separated from other [illegible] clearly defined form. These adjacent areas indicate the [illegible] may be present in sections from the same gland. Many types may be present in one gland. Yet the predominant picture is usually of one single type.

and took thirty-six hours to reach +50 again. In 2 other cases there was no increase of basal following massage. No control was made in these cases by massage elsewhere.

This lymphatic hyperplasia is not of the type represented by the lymphocytic infiltrations which are associated with in-

flammatory reaction, but is a collection of lymphoid germinal areas with foci of lymphocyte production, rather than lymphocyte invasion

These lymphatic germinal areas present some problems upon which further data should be given The question as to the relation of these lymphocytic areas to the regeneration and to

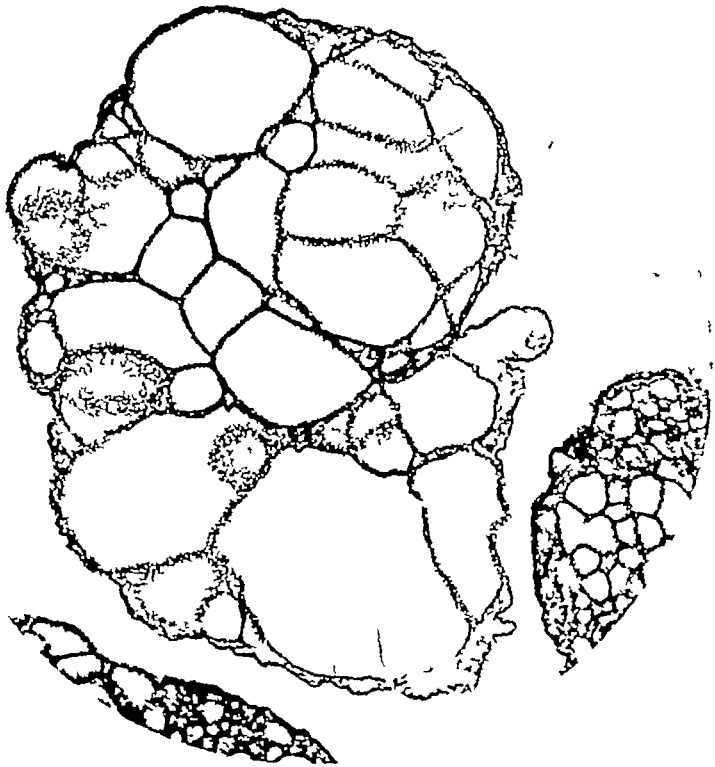

Fig 91

the cyclic structural changes in the gland should be answered The fate and future of the lymphocytes produced by these areas should be of interest Maximow has recently shown the growth capacity of the lymphocyte, and, in spite of the limitation imposed by the germ-layer theory, the anatomic position of this lymphatic group might prove of further interest

It may be seen that the structure of the thyroid in these

cases of toxic goiter varies quite remarkably in anatomic pattern The cases present a clinical similarity of sufficient clearness to be identical except for the cases of exophthalmic goiter, and in these the exophthalmos remains the clearest outstanding point of difference

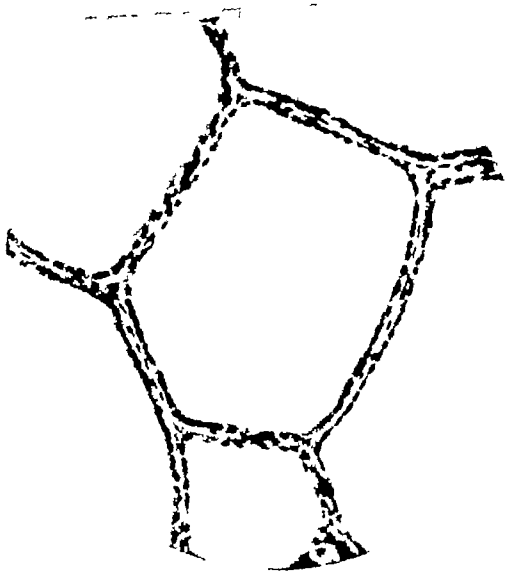

Fig 92

Figs. 91 and 92.—Showing a lobule of the more common type of structure. Large alveoli filled with colloid flattened lining cells and little evidence of hyperplastic change The high magnification shows the characters of the cells lining the acini. No cellular structure between acini the acini being directly contiguous to one another

The problem of the etiology of the clinical syndrome associated with pathologic changes in the thyroid remains an open problem of interest Despite the recent work in this direction, the major problem of function of the gland remains unanswered

The isolation from the thyroid gland of an iodin compound, thyroxin, by Kandall and its further identification by Harrington throw only a partial light on the function of the thyroid gland itself It is possible that the thyroid gland produces no compound similar to this in its normal cycle of physiologic activity

Fig 93 —Showing type of epithelium lining alveolus, and cushion formation beginning papillary ingrowth in thyroid, showing moderate gland of hyperplasia with incomplete loss of alveolar arrangement

The changes in structure of the gland should reflect the demand of its function and further knowledge concerning the significance of the structural change is much to be desired

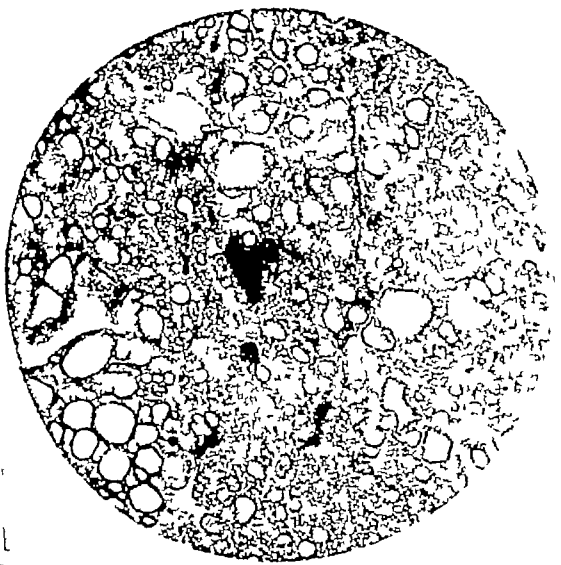

Fig. 94.—Lymphatic invasion of thyroid of marked cellular structure with many well-defined acini and cellular interacinar structure of solid pattern

Fig 95

Figs. 95 96.—Showing evidence of hyperplasia and hypertrophy with partial retention of alveolar structure.

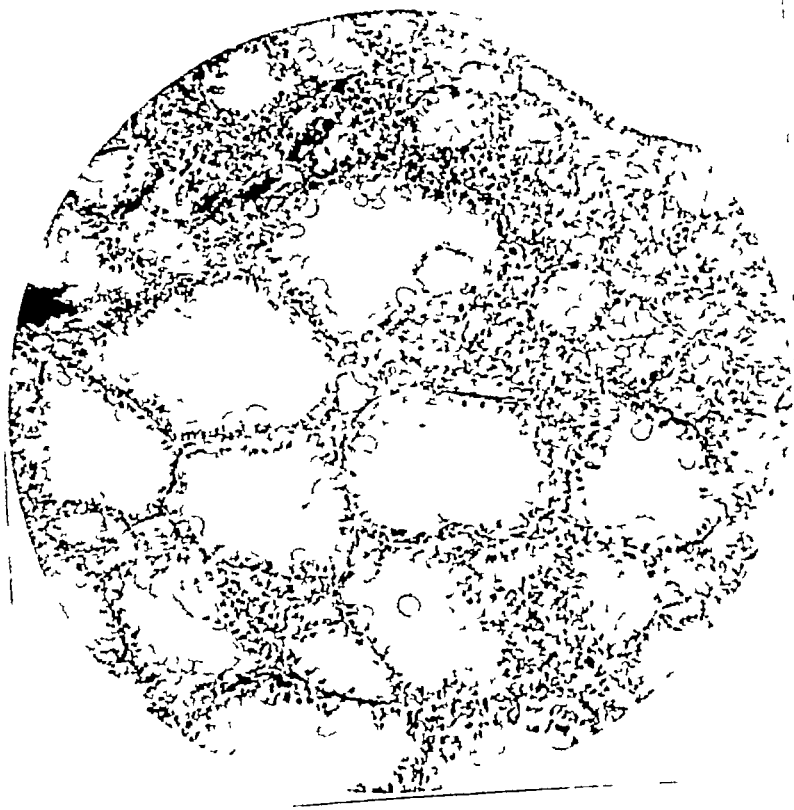

Fig 97

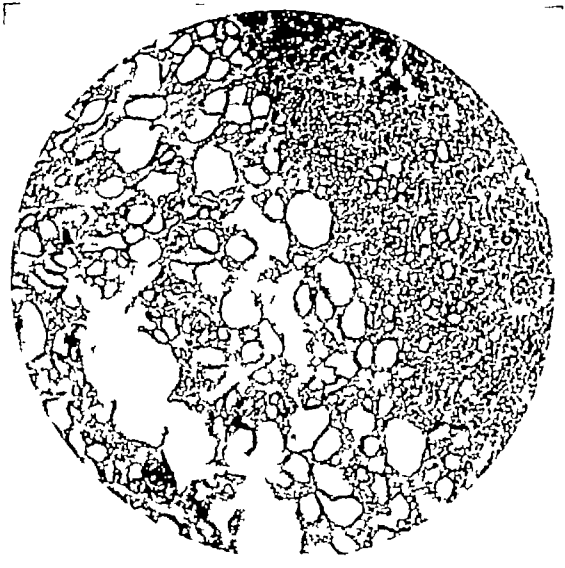

Fig 98

Figs. 97 98.—Showing evidence of hyperplasia and hypertrophy with partial retention of alveolar structure.